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VOLUME 26
1931

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

Medical
Mahr

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Archives of Neurology and Psychiatry

VOLUME 26 JULY, 1931 NUMBER 1

EFFECTS OF FRAGMENTARY AND COMPLETE EXTIRPATION OF THE CEREBELLUM IN THE CAT

A PRELIMINARY REPORT ON CEREBELLAR LOCALIZATION *

ARMANDO FERRARO, M.D.
AND
LEO M. DAVIDOFF, M.D.
NEW YORK

It is well known that while a number of authors oppose the conception of definite localization in the cerebellum, others still believe that such localization exists, and that definite centers are present for the regulation of the finest movements of the eyes, head, neck, trunk, legs, etc. This controversy has again come to the foreground since the recent revival of the work of Horsley and Clarke by Aubrey Mussen, who, by means of electrical stimulation, has reached the conclusion that well localized centers are to be recognized in the cerebellum, while Riley, from a study of comparative anatomy, has reached an essentially opposite opinion.

Our approach to this complex problem has been by means of extirpation of different parts of the cerebellum as well as of the whole organ and by studying the resulting impairment of functions. Other authors, for example, Luciani, Ferrier, Thomas, van Rijnberk, Rothman, Rossi and Rademaker, have made brilliant contributions by the use of this method. It was our hope, however, that guided by the phylogenetic rather than by the purely morphologic divisions of the cerebellum in our extirpations, making use of modern neurosurgical technic in our operating, and finally analyzing the defective function of our animals by means of slow motion pictures, we might glean some new facts toward an understanding of the function of, as well as the localization of function in, the cerebellum.

The animals operated on were also used to study the action of bulbocapnine on the central nervous sytem when deprived of part or of the whole of the cerebellum. This work will form the subject of a later paper.

* Submitted for publication, Aug. 28, 1930.

* From the Department of Neuropathology of the New York State Psychiatric Institute and Hospital.

* Read at a meeting of the New York Neurological Society, Feb. 4, 1930 (the presentation consisted of moving pictures of the animals operated on).

Finally, another aim of our work is the study of the anatomic correlation between the various areas of the cerebellum and the various pontile nuclei. For this purpose all the material of our operative series has been cut serially, and a particular study will be made of the various pontile nuclei as well as of the olivary nuclei.

The series of animals operated on by Dr. Davidoff numbered up to fifty. Our present report will deal, however, with only sixteen, representing the various types of removal. Usually, duplicates of the same operation were made in order to confirm previous results. The lesions experimentally produced have always been confirmed at autopsy, and some of the brains have already been serially sectioned for the study of both the extent of the lesion and its repercussion on the pontile nuclei. To point out the experimental lesions, we have made use of the folial pattern scheme as given by Riley, with only slight modification of the proportions between the lateral lobes and the vermis.

For the nomenclature, we have made use of the one given by Bolk, who divides the cerebellum into a lobus anterior, a lobus simplex and a lobus posterior, the lobus anterior and the lobus simplex being separated by the fissura primaria. To the lobus anterior belong the lingula, the lobus centralis and the culmen; to the lobus posterior belong the declivus, the tuber, the folium, the pyramis, the uvula, the nodulus along the midline, and crus I and crus II of the lobus ansiformis, the lobulus paramedianus, the paraflocculus and the flocculus along the lateral aspect.

Riley recently modified Bolk's nomenclature, especially in regard to the lobus anterior, where he distinguishes between the vermal and the lateral portions by adding to the number of the lobule the qualification of lateralis or vermalis. Another distinction made by Riley is the division of lobus C^2 of the vermis into a lobus C^2 presulcal and C^2 postsulcal, separated by the sulcus intercruralis (great horizontal fissure).

Although we have not made exclusive use of Riley's changes, we have mentioned them in correlation with the old nomenclature. We express, however, the hope that Riley's suggestions will be universally adopted, thus greatly simplifying the complicated cerebellar nomenclature.

For the purpose of comparison, we have drawn a scheme in which the old classic and the recent nomenclatures are shown (fig. 1.).

REMOVAL OF PARTS OF OR OF THE WHOLE LATERAL LOBE

CAT 1.—The animal was operated on for the removal of the right lobus simplex. At autopsy it was found that the removal destroyed four fifths of the extension of the right lobus simplex, respecting the very anterior part of it and extending caudad into the declivium of the vermis, of which a small portion was removed (fig. 2). Following the operation the animal showed no clinical signs

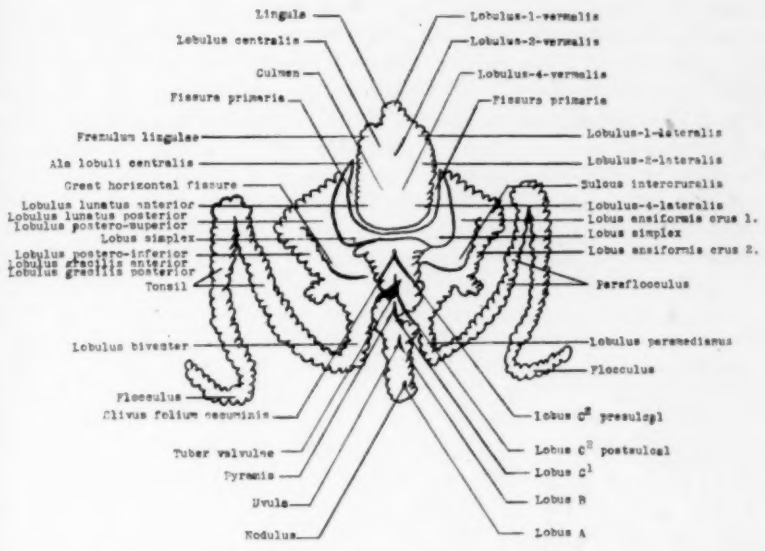

Fig. 1.—Diagram of the cerebellum to compare the old descriptive nomenclature (left side) with the modern nomenclature based on ontogenesis—Bolk nomenclature modified by Riley (right side).

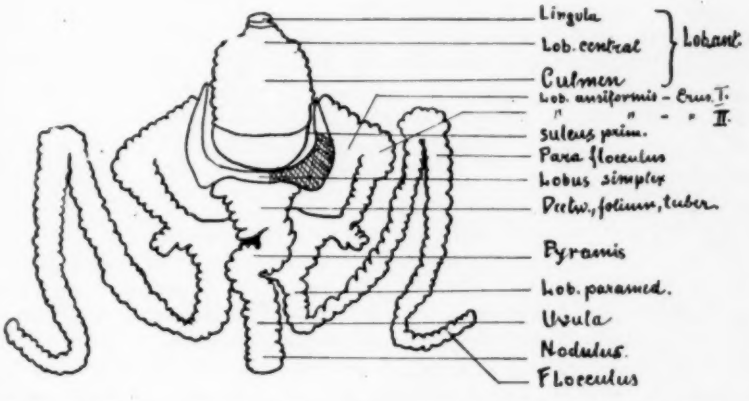

Fig. 2.—Diagram showing the removal of the right lobus simplex. The area removed is demonstrated by ink network.

of any importance. Gait, posture and reflexes were normal. No apparent tremor of the head nor dysmetria of the neck muscles was present. A year after the operation, just before autopsy, the animal behaved normally, and no clinical signs of cerebellar disturbances were noticeable. The cat could walk, run and jump like a normal animal. The righting reflexes and postural reflexes appeared to be normal.

Cat 2.—The animal was operated on for the removal of the posterior portion of the lobulus paramedianus of the right side. At autopsy it was shown that the lesion involved the posterior half of the lobulus (fig. 3), respecting the anterior portion and its transition into the crus II of the lobus ansiformis. Following operation the animal recovered in a few days, and it was noticed that it presented a certain amount of asynergia (dysmetria) of the hind right leg. The foreleg was normal, and nothing was noticeable on the left side.

A year after the operation, at the time of death, the animal still presented asynergia of the right hind leg. No disturbed muscular tonus was noticeable in the affected leg.

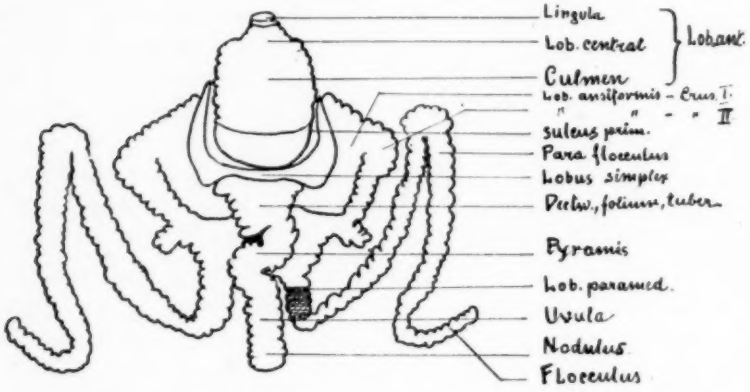

Fig. 3.—Diagram showing the removal of the posterior portion of the right lobulus paramedianus.

Cat 3.—The animal was operated on for the removal of a slightly larger portion of the right lobulus paramedianus. At autopsy it was shown that the lesions involved a larger portion of the lobulus paramedianus than in cat 2, the lesion extending more anteriorly (fig. 4).

The animal presented asynergia of the right hind leg with the addition of a slight asynergia in the front leg of the same side.

Cat 4.—The animal was operated on for the removal of the whole right lobulus paramedianus. At autopsy, performed a year later, the removal was seen to involve the whole lobulus paramedianus and its transition into the posterior half of crus II of the lobus ansiformis (fig. 5). Soon after the operation, the animal showed asynergia in both front and hind right legs. The asynergia consisted in marked hypermetria of both legs, but was more pronounced in the front one. In walking, the animal raised its front leg higher than normally, overextending and overflexing the various segments of the leg, the movement resulting in what might be called a "goose step." Righting reflexes, running and jumping appeared normal.

CAT 5.—The animal was operated on for the removal of the right crus I. At autopsy the lesion involved the whole of crus I plus a slight portion of the lobus simplex (fig. 6). Clinically, the animal disclosed dysmetria of both front and hind homolateral legs with predominance of the symptoms in the fore leg in which a goose step was pronounced.

CAT 6.—The animal was operated on for the removal of the left crus I and crus II of the lobus ansiformis. At autopsy the lesions appeared to involve the

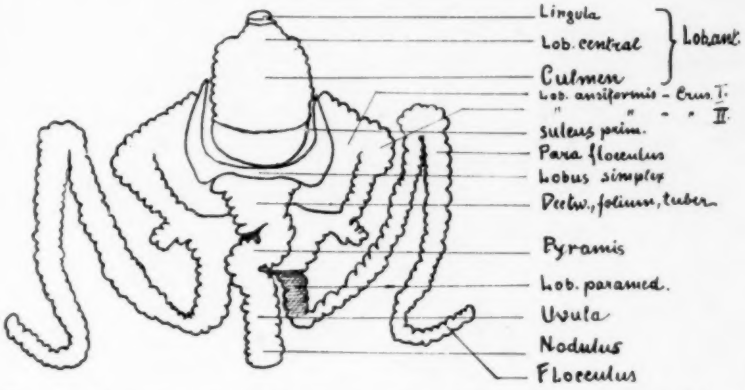

Fig. 4.—Diagram showing the removal of a slightly larger portion of the right lobulus paramedianus than that shown in figure 3.

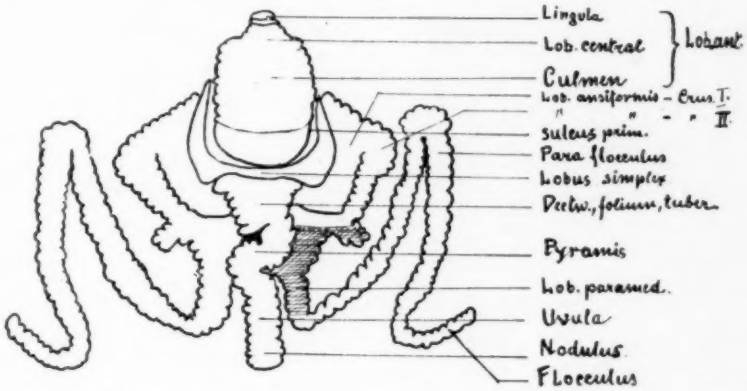

Fig. 5.—Diagram showing the removal of the whole right lobulus paramedianus.

whole of crus I and II and a small area of the lobulus paramedianus. The lobus simplex of the same side was also removed (fig. 7). Clinically, the animal showed a tendency to fall over toward the left, and there was increased tonus on the same side. Gradually these symptoms disappeared, and the only noticeable residue was the definite dysmetria of both the hind and front left legs. No involvement of the head or of the neck was apparent.

Cat 7.—The animal was operated on for the removal of the entire right lateral lobe including the lobus simplex, crus I and crus II of the lobus ansiformis, the lobulus paramedianus, the paraflocculus and the flocculus. At autopsy, performed a year later, it was found that the whole lateral lobe was absent, with the exception of a small piece of tissue representing a disconnected part of the paraflocculus (fig. 8). Following the operation, the animal showed a marked hypertonus with both right legs overextending under the slightest stimulus. The rigidity thus

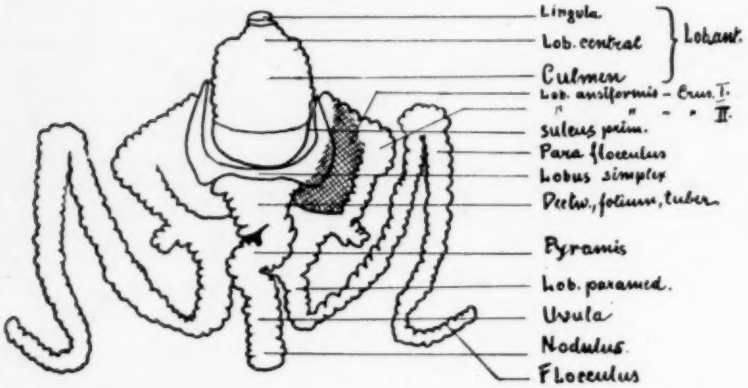

Fig. 6.—Diagram showing the removal of the right crus I and a slight portion of the homolateral lobus simplex.

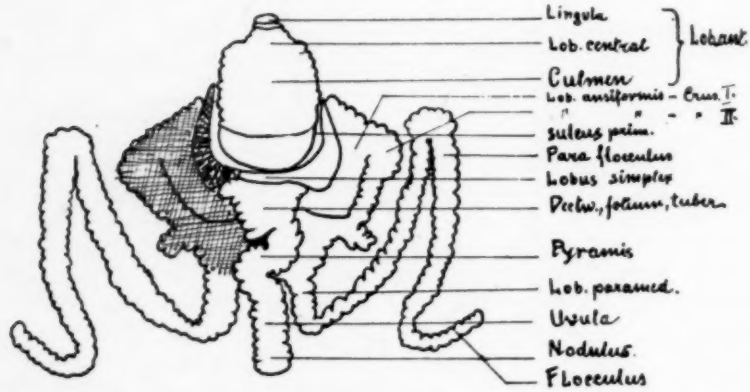

Fig. 7.—Diagram showing the removal of the left crus I and crus II in addition to the lobus simplex of the same side.

resulting was definite and well appreciable in the performance of passive movements. In the first month following operation, the animal had a tendency to fall toward the right. In walking, it kept a wide base with the front and hind right legs. There was overextension of the right front leg (dysmetria) resulting in the already mentioned "goose step" and definite dysmetria of the right hind leg.

Several months after the operation, compensation had occurred, and the animal no longer showed the tendency to fall toward the right. Eleven months after

the operation, the only clinical manifestation appreciable was a definite asynergia of both anterior and posterior right legs with a predominance of the dysmetria on the front one, still showing a definite "goose step."

No disturbed tonus was noticeable in any of the four extremities nor were the righting reflexes disturbed.

CAT 8.—The same operation was undertaken as in cat 6. At autopsy it was found, however, that the lateral lobe was completely removed with no apparent injury to the vermis (fig. 9). Immediately following the operation, the animal

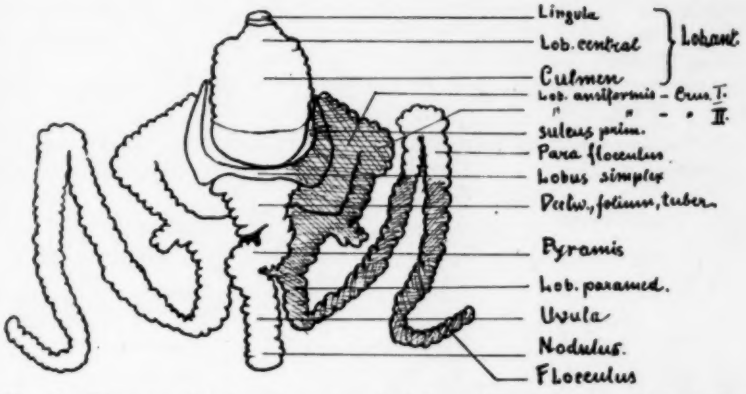

Fig. 8.—Diagram showing the removal of the entire right lateral lobe including the lobus simplex, crus I and crus II, the lobulus paramedianus, the paraflocculus and the flocculus.

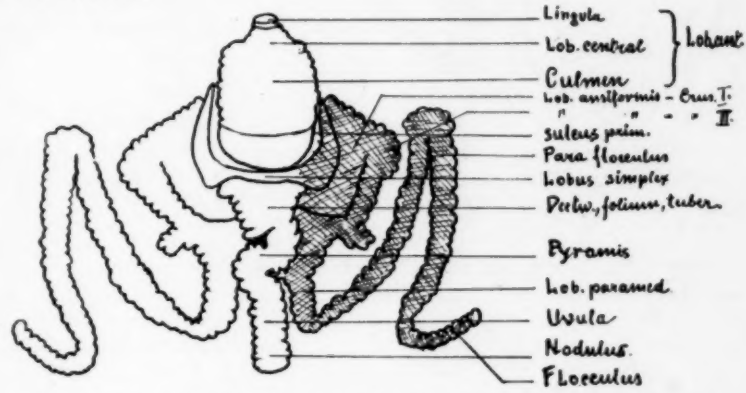

Fig. 9.—Diagram showing extension of the removal as in figure 8.

showed rigidity in extension on the side operated on. A few weeks later the animal showed a definite tendency to fall toward the side of the removal, but gradually this tendency to fall disappeared, and the only remaining symptoms were represented by slight deviation of the tail toward the right side and a wide base in walking due to the lateralization of both front and hind right legs. Only occasionally a false step was noticeable leading to a tendency to sway toward the right. The removal of the paraflocculus, which was intact in the previous animal, seemed to result in a deviation of the tail toward the side operated on.

Cat 9.—The animal was operated on for the removal of the whole lateral lobe. At autopsy, performed fifteen months later, it was found that the whole lateral lobe was removed with the exception of the most frontal portion of the paraflocculus and of the flocculus. It was found, besides, that a superficial lesion of the vermis was produced involving the most lateral parts of the lingula, lobus centralis and culmen (lobules I, II and IV vermalis of Riley) as well as the most lateral part of the tuber (lobus C^2 presulcal and postsulcal) (fig. 10). Following the operation the animal showed a definite tendency to fall toward the right, accompanied by a tendency to rotate toward the right. The tail was always kept in a markedly curled position turned toward the right, which led us to designate the cat as "the monkey cat." The falling to the right gradually disappeared, and the only symptoms persisting even fifteen months after the operation were an oblique gait toward the right and the curling of the tail. No disturbances in the tonus or in the righting reflexes were observed.

Cat 10.—The operation was performed to remove both the lateral lobes. At the autopsy, carried out a year later, it was found that on the right side, the

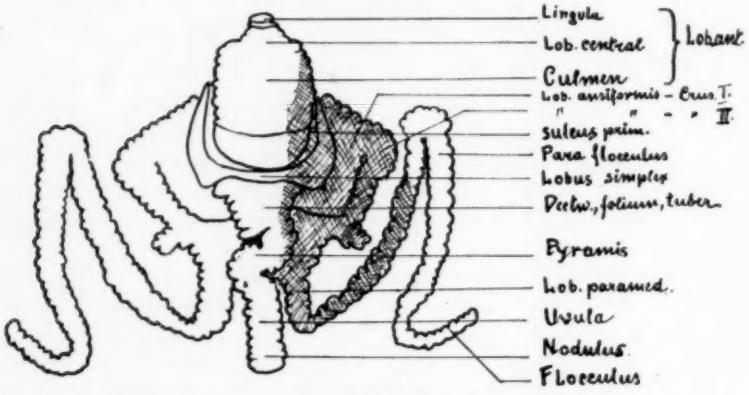

Fig. 10.—Diagram showing removal of the right lobus simplex, lobus ansiformis, lobulus paramedianus and paraflocculus. There was an additional removal of the most lateral portion of the vermis (right side).

lobus simplex, crus I and crus II of the lobus ansiformis were entirely removed and that the lobulus paramedianus and the caudal part of the paraflocculus were intact. The flocculus also was present, although disconnected from the rest of the organ. On the left side, the lobus simplex, crus I and crus II of the lobus ansiformis and the dorsal half of the lobulus paramedianus were entirely removed (fig. 11). The paraflocculus and flocculus were present.

A few weeks after the operation, the animal staggered toward either side with a tendency to fall, especially to the left. This symptom gradually disappeared, and a few months later the only symptom present was a certain amount of asynergia (dysmetria) of both the legs on the right side. On the left side the dysmetria was more pronounced, and the front leg showed a definite "goose step." No disturbances in the righting reflexes or in the muscular tonus of the extremities were noticeable.

Cat 11.—This animal was operated on for the removal of the whole lobus simplex of both the lobi ansiformes and of the corresponding portion of the vermis represented mainly by the vermal portion of the lobus simplex, the decliv-

ium, the folium and the tuber (fig. 12). This animal was still alive eighteen months after the operation. Immediately following the operation it presented hypertonic phenomena involving the four extremities. The animal was unable to walk because of a marked ataxia of the posterior part of its body. In the attempt to rise, an overextension of the musculature of the neck and of the upper part of the trunk occurred, leading to a retraction of the neck and body, the animal then falling backward. All the attempts to move forward were handicapped by

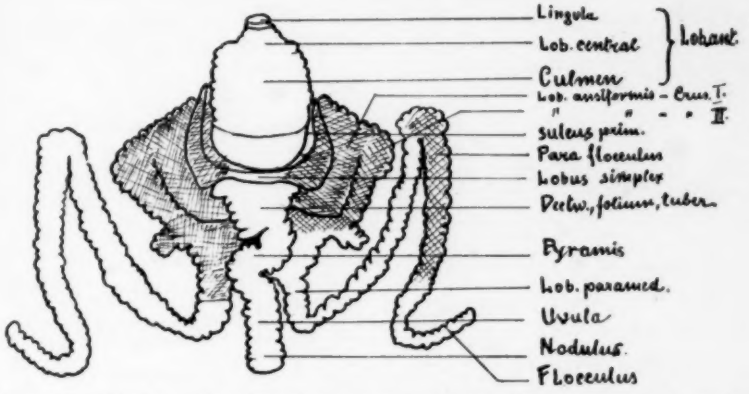

Fig. 11.—Diagram showing the removal of the whole lateral lobe with the exception of the flocculus and paraflocculus of the left side and of the lobus simplex, lobus ansiformis, and a portion of the paraflocculus of the right side.

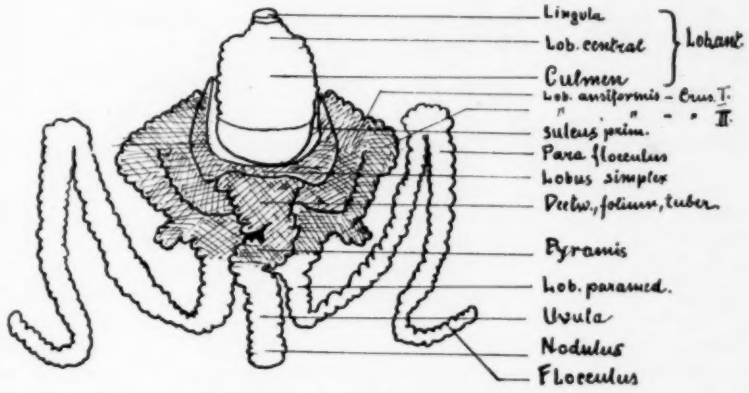

Fig. 12.—Diagram showing the removal of both lobi simplices, lobi ansiformes, and the corresponding middle portion of the vermis (declivium, folium, and tuber).

the apparent inertia of the posterior part of the body. In the early weeks following operation there was a definite tremor of the head and definite hammering of the head in the attempts at feeding. A few months later the animal was able to walk, but it showed a definite tendency to fall toward either side and a swinging of the body from left to right. In walking, the foot base was much widened in both the anterior and posterior extremities.

In the numerous falls it often looked as if the fall, either to the right or to the left, was provoked by the inability of the animal to coordinate the movement

of the posterior portion of the trunk and of the pelvic girdle. The extreme swinging of the body toward the right or toward the left seemed responsible for the fall of the animal on the corresponding side.

Sixteen months after the operation, the symptoms already mentioned, although less in intensity, were fundamentally the same. The animal succeeded in walking, but still maintained a very wide base with both front and hind legs. The hammering of the head had disappeared, and no definite intention tremor was noticeable. The muscle tonus, the deep reflexes and the righting reflexes appeared normal. There was an increase in postural activities, the animal always trying to reestablish quickly a disturbed postural attitude. In walking there also seemed to be an increased sustaining tonus.

REMOVAL OF PARTS OF OR THE WHOLE VERMIS

CAT 12.—The operation was performed to remove a small portion of the lobus anterior consisting of part of the culmen (lobulus IV vermalis of Riley)

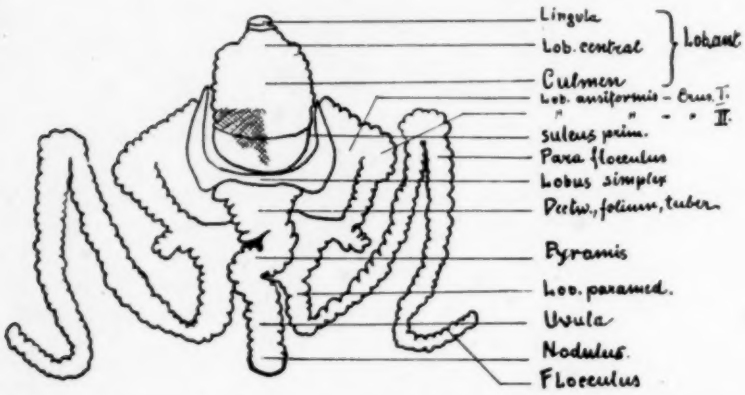

Fig. 13.—Diagram showing the removal of a portion of the left lobus anterior.

and part of the lobulus lunatus anterior (lobulus IV lateralis) on the left side. At autopsy, the removal was found to involve exactly the previously mentioned structures (fig. 13). A week after the operation the animal could freely move around, run and jump, the only clinical manifestation being represented by a certain amount of dysmetria of the left foreleg and a certain amount of shaking of the head, especially when at rest. No other changes occurred in the following months. At the time of autopsy, twelve months later, the symptoms were exactly the same as those reported a week after the operation. Probably now and then the dysmetria of the anterior legs was more pronounced and the shaking of the head was more obvious.

CAT 13.—The operation was performed to remove the superficial portion of the vermis along the midline. Autopsy, performed a year later, showed that the removal involved a narrow and superficial band of the vermis, which extended from the culmen to the tuber inclusive (lobulus IV vermalis, C^2 presulcal, and part of C^2 postsulcal) (fig. 14). Soon after the operation, the animal was unable to keep a standing position, falling indiscriminately toward the right or the left.

Gradually, compensation occurred and the animal was able to walk although with a widened base. In the attempt to climb stairs, the animal often fell backward. Ter months after the operation there remained only an uncertainty in gait. This consisted in asynergia of the trunk and of the legs resulting in a slight swinging of the body from left to right or vice versa and a gait with widened base of the fore and hind legs. No actual falling occurred. Now and then a shaking of the head was noticeable.

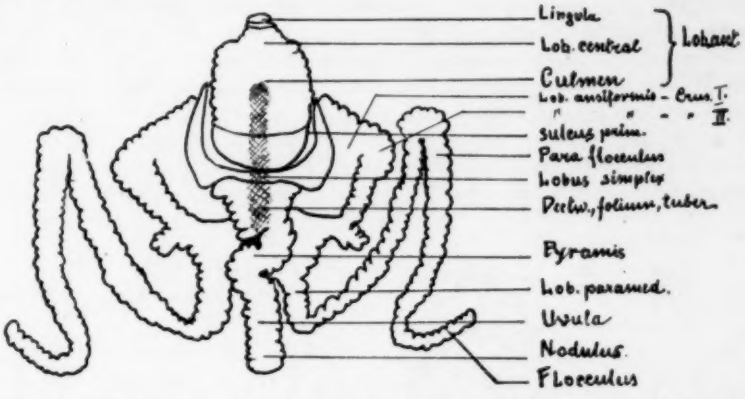

Fig. 14.—Diagram showing the removal of a superficial portion of the vermis along the midline.

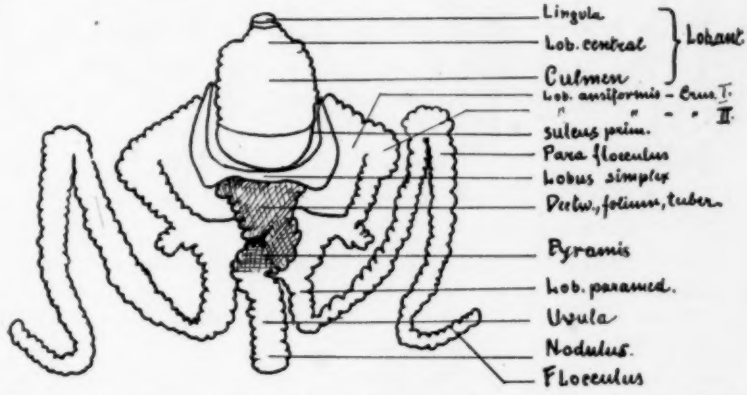

Fig. 15.—Diagram showing the removal of the declivium, folium, tuber, and anterior portion of the pyramis.

CAT 14.—The operation was performed to remove a large portion of the vermis posterior to the fissura primaria. At autopsy, performed a year later, it was found that the vermal portion of the lobus simplex, the declivium, the folium, the tuber and the anterior part of the pyramis (lobus C^2 presulcal and postsulcal and lobus C^1 vermalis) were removed (fig. 15). Immediately after the operation, the animal was unable to stand or walk and disclosed a definite hypertonus of the four extremities in the various attempts at reacquiring a normal posture. Several weeks later, when the animal began to walk, it was found that the most difficult task for him was to coordinate the hind legs and the posterior portion

of his trunk. The latter showed considerable swinging from left to right or vice versa, producing, when extremely pronounced, the fall of the animal. The animal walked with a much widened base, and the gait was dysmetric. There was not, however, the typical "goose step" that we noticed in cases following the removal of the lateral lobes. The asynergia seemed limited to the muscles of the trunk. The animal was killed a few months after the operation, and no chance was given to evaluate the amount of compensation that might have occurred later.

Cat 15.—The operation was performed in order to remove the whole vermis leaving as intact as possible both lateral lobes (fig. 16). Following the operation, the animal was unable to make any use of the hind legs in walking, and it looked as if a motor paralysis had occurred. The doubt was soon cleared up, however, when a few weeks afterward it was noted that while lying on the side he could utilize the hind limbs in scratching himself most actively and accurately. It appeared clear that the incapacity to make a correct use of the hind legs in walking was related to the incapacity in coordinating the movement of these legs

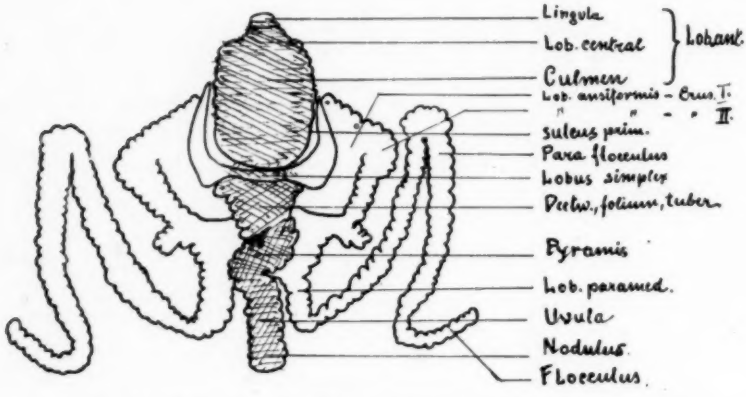

Fig. 16.—Diagram showing the removal of the whole vermis.

with the movement of the trunk. In the early period following the operation, this animal also disclosed hyperextension of the forelegs in various attempts at movement and also showed tremor and the so-called "hammering" of the head.

Seven months after the operation, the animal was able to walk, but the gait was incoordinated, the swinging of the body pronounced and the falling toward either side frequent. The animal showed definite asthenia, becoming tired rapidly after each attempt to walk. Even a year after the operation, though the animal had improved considerably and was able to walk certain distances without falling, it was still noticeable that the main difficulties with the gait lay in the incoordination of the posterior portion of the trunk that still swung considerably from one side to the other, thus requiring a wide base posteriorly.

No disturbed tonus was noticeable at that time, and the righting reflexes appeared normal. It was interesting to note the marked overactivity of the tonus of support (Stutztonus). The animal was still alive at the time this paper was written.

REMOVAL OF THE WHOLE CEREBELLUM

CAT 16.—The operation was performed in order to remove the cerebellum in toto. Autopsy, performed two months later, showed that the whole cerebellum was removed with the exception of the paraflocculus and the flocculus of the right side (fig. 17). Immediately following the operation the animal was unable to stand or to walk, and disclosed a marked hypertonicity of the forelegs. There was a considerable tremor and hammering of the head.

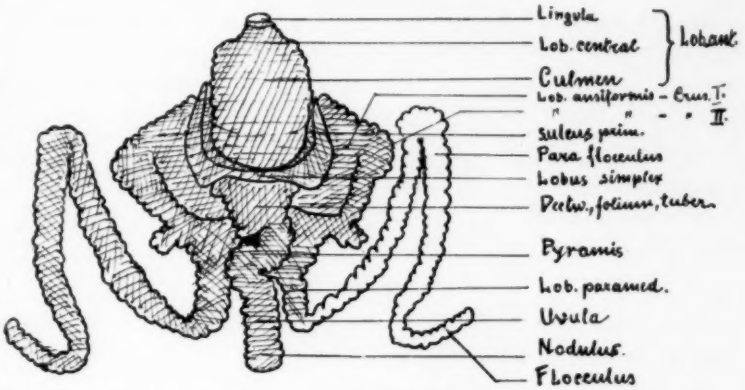

Fig. 17.—Diagram showing removal of the whole cerebellum, with the exception of the right flocculus and paraflocculus.

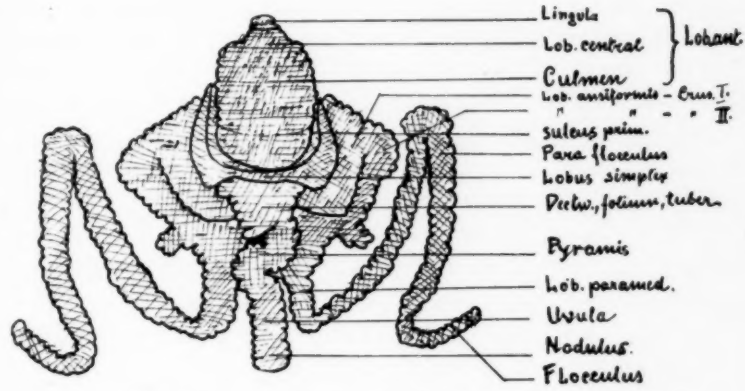

Fig. 18.—Diagram showing removal of the whole cerebellum.

In the two months following the operation, no marked improvement took place, the animal being unable to walk, especially because of marked incoordination of the hind legs and of the trunk. In this case also the animal looked as though it were paralyzed, but, as in the previous case, it was able to make separate use of the hind legs. Even when lying down, considerable movement of the posterior part of the body took place, thus generating the fall of the animal toward the right or toward the left, especially in the attempts to correct an improper position.

The only attempt at walking made by the animal consisted in crawling with the fore legs and at the same time dragging the posterior portion along the floor.

CAT 17.—The operation was performed with the intention of removing the whole cerebellum, which was successfully achieved, the entire cerebellum being removed almost intact (fig. 18). The animal was still alive sixteen months after the operation.

The various early reactions following the operation were exactly comparable to those in cat 15. It was only eight months after the operation that an attempt to walk by making use of the fore legs was noticeable. The animal dragged the hind legs in attempts at walking, although he was capable of making separate use of the legs in individual movements, such as scratching himself.

Ten months after the operation, the animal was able now and then to make use of the hind legs with a certain amount of coordination; very often swinging of the body would produce its fall to either left or right. Sixteen months after the operation, much better coordinate movements were noticeable, and the animal was capable now and then of standing on its four feet and of walking a few steps, although the posterior part of the body was closer to the ground than the anterior part.

COMMENTS

In order to analyze the results of our investigation, we must reiterate some of our observations.

In cat 1 the so-called lobus simplex alone was removed. Riley recently proposed to consider the lobus simplex not as a separate entity, but as a part of the anterior lobe located behind the sulcus primarius. The fact that in cat 1 no clinical manifestations followed the removal of the lobus simplex seems to uphold Riley's conception against the one of Bolk, van Rjinberk, Ingvar and Aubrey Mussen who attach a localizing and phylogenetic importance to the lobus simplex.

Cat 2 showed dysmetria of the hind right leg following a removal of a small area of the posterior portion of the lobulus paramedianus. No appreciable symptoms were apparent in the front legs.

Cat 3 showed slight dysmetria of both fore and hind right legs, although less marked in the fore leg, following the removal of a slightly larger portion of the same lobulus toward the anterior portion.

Cat 4 showed definite dysmetria of both the fore and the hind right leg following the removal of the whole lobulus paramedianus and a small portion of crus II of the lobus ansiformis of the same side.

It follows, then, that the lobulus paramedianus seems to control in a special way the synergia of both fore and hind legs. In some animals the synergia of the hind leg seems to be controlled more by the posterior portion of this lobulus paramedianus, while in others the posterior portion controls both the fore and hind homolateral legs. It is evident then that the lobulus paramedianus controls as a whole the

synergia of both the homolateral legs and that a definite boundary between the territory controlling the posterior and the anterior legs is not always possible to demonstrate.

From cat 5, crus I of the lobus ansiformis and a small additional part of the lobus simplex had been removed. In this animal there was also a dysmetria of both fore and hind homolateral legs, but more pronounced in the anterior one in which a goose step was present.

From cat 6, crus I and crus II of the left lobus ansiformis had been removed. The animal disclosed asynergia of both front and hind homolateral legs, more pronounced in the front legs.

It follows, then, that in crus I and crus II there is a controlling center for the synergia of both anterior and posterior legs, but that no division seems to exist between the center controlling the front legs from the center controlling the hind legs, although it seems that the lesion of crus I while involving both front and hind legs affects the front one more severely.

In cats 7 and 8, in addition to the removal of the lobulus paramedianus, the lobus simplex, crus I and crus II of the lobus ansiformis, the paraflocculus and the flocculus were removed.

In the early stages following the operation there was definite falling toward the operated side. This falling gradually disappeared and was replaced several months after the operation by merely a tendency to fall, with compensatory movements successfully preventing an actual fall. The difference in the symptoms, therefore, between the removal of the lobulus paramedianus and the lobus ansiformis (crus I and crus II) alone, and the removal of the entire lateral lobe consists in the fact that in the former case no fall was noticeable. That is, the permanent result in the case of the removal of either the lobulus paramedianus or the lobus ansiformis is the dysmetria of the homolateral legs, whereas, the removal of the flocculus and paraflocculus together with the lobulus paramedianus and ansiformis results in falling toward the homolateral side which is eventually compensated for by widening of the base and a set of reflex movements of defense or protection that prevents the actual fall.

In cat 10 the lobus simplex, plus the lobus ansiformis on the right side and the lobus simplex, plus the lobus ansiformis and the lobulus paramedianus on the left side were removed.. In this animal the left side showed definitely more marked dysmetria of the legs, especially of the foreleg (goose step) thus confirming the conclusion that the lobulus paramedianus is concerned with the synergia of the fore and hind legs.

Considering now cats 7, 8 and 9, it will be observed that in these three animals, besides the removal of the lobus simplex, the lobus ansiformis and the lobulus paramedianus, the paraflocculus was removed. Besides the falling toward the side operated on and the dysmetria of both fore and hind legs, these animals showed a more or less marked curling of the tail toward the side on which operation was performed.

It seems justifiable then to connect the removal of the paraflocculus with the symptoms occurring in the tail.

In cat 12 there was removed a small portion of the lobus anterior (lobulus IV vermalis and lateralis). The animal showed definite tremor of the head, even when resting, We may safely deduce, then, that these areas control the synergia of the muscles of the neck. There was also a certain amount of dysmetria of the homolateral fore leg, thus pointing to the possibility that this area contains also an accessory center for the synergia of the fore leg.

In cat 14, following the removal of the medial portion of the vermis corresponding to the declivium, folium, tuber and part of the pyramis (lobus C^2 presulcal and postsulcal and lobus C^1 vermalis) the animal showed considerable swinging of the body from one side to the other, thus allowing us to consider this portion of the vermis as the controlling center for the synergia of the muscles of the trunk, especially in the shoulder girdle.

Cat 15, in which the whole vermis was removed, showed considerable swinging of the body, more pronounced over the pelvic girdle. In this animal, in addition to the portion of the vermis removed in cat 14, the rest of the pyramis, uvula and nodulus were removed. The involvement of the muscles of the trunk and of the pelvic girdle was so pronounced as to impair the standing position of the animal.

We feel justified, then, in concluding that in the vermis resides the control for the synergic movement of the neck and trunk. While the controlling center for the muscles of the neck is localized in the anterior portion of the vermis, lobulus lunatus anterior and culmen (lobulus IV lateralis and vermalis), the controlling center for the synergia of the muscles of the anterior portion of the trunk, especially of the shoulder girdle, is localized in the middle portion of the vermis, that is, the declivium, folium, tuber and possibly the anterior portion of the pyramis (C^2 presulcal and postsulcal, C^1 vermalis), and the controlling center for the synergia of the muscles of the posterior portion of the trunk, especially of the pelvic girdle, is localized in the posterior portion of the vermis, mainly the pyramis, the uvula and the nodulus (C^1 vermalis, lobus B and lobus A).

The importance of the posterior portion of the vermis in the control of the synergia of the pelvic girdle must be considerable if one considers that following the removal of the whole vermis, either separately or in combination with the lateral lobes (total decerebellation), the animal is unable for several months to keep an upright position because of the extremely pronounced swinging of the pelvis from side to side.

Nearly all of our extirpations have been very deep and not limited to the cortical layers only. This fact is important because of the involvement of the nuclei in our cases, a circumstance which may modify the result of the removal according to Simonelli and Mussen.

COMPARISON WITH OTHER INVESTIGATIONS

According to Bilancioni,[1] in 1749, a physician from Rimini, Giovanni Bianchi, on the basis of a clinical observation attempted to formulate some principles of cerebellar localization, concluding that each hemisphere of the cerebellum has a control that is dominantly homolateral. Rolando,[2] in his rudimentary experiments, reached the same conclusion. In 1876, Ferrier [3] found that removal of the anterior portion of the vermis produced a tendency of the animal to fall forward, while removal of the posterior portion of the vermis caused falling backward. In 1897, Thomas [4] found that in dogs there is a certain relationship between the vermis and the musculature of the fore trunk and legs and between the cerebellar hemispheres and the muscles of the hind trunk and legs.

Bolk,[5] in 1904, was the first author who attempted a systematic localization in the cerebellum by the study of comparative anatomy. From the results of his investigation he designated the lobus anterior as the center for the coordination of the muscles of the head, the lobus simplex as the center for the coordination of the muscles of the neck, crus I as the center for the coordination of the anterior leg, crus II as the center for the coordination of the posterior leg, and the formatio vermicularis as the center for the coordination of the musculature of the tail. In the lobulus paramedianus Bolk placed the center for the regulation of the two halves of the trunk.

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1. Bilancioni, G., quoted by Luciani: Fisologia dell'uomo, ed. 5, Milan, Società Editrice Libraria Milanese, 1919, vol. 3, p. 600.

2. Rolando, L., quoted by Luciani (footnote 1).

3. Ferrier, D.: Functions of the Brain, London, Smith Elder & Company 1896.

4. Thomas, A.: Le cervelet, étude anatomique, clinique et physiologique, Paris, 1897.

5. Bolk, B.: Das cerebellum der Säugetiere, Jena, Gustav Fischer, 1906.

Bolk's scheme of localization was later experimentally confirmed by detailed work by van Rijnberk,[6] Rothmann,[7] Thomas and Durupt,[8] Pagano,[9] Luna,[10] Rossi,[11] Simonelli,[12] and Ingvar.[13]

Among the conclusions of van Rijnberk's work, which was carried on in Luciani's laboratory in Rome, the following are of interest: 1. The removal of the lobus simplex is followed almost exclusively by tremor of the head due to ataxia of the muscles of the neck. 2. The removal of crus I of the lobus ansiformis is followed by considerable dysmetria in the fore leg (rooster step). 3. The removal of crus II, especially in the transitional area between these lobules and the lobulus paramedianus is followed by no definite dynamic phenomena except simple asthenia of the muscles of the hind leg of the same side, which gives way under the weight of the animal's body. 4. Removal of the lobulus paramedianus is followed by rotation of the animal along its longitudinal axis toward the side on which operation was performed.

Rothmann's experiments disclosed that removal of the entire lobus anterior resulted in involvement of the lower jaw and larynx, and curving of the posterior portion of the body, accompanied by severe ataxia of the hind legs. Lesions of the upper portion of the lobus anterior are followed by dysmetria and rigidity of the head. Lesions of crus I resulted in dysmetria of the front leg, while lesions of crus II resulted in dysmetria of the posterior leg. If the lesion of crus I and crus II involved the lateral portion of the lobuli, the movement of adduction was mainly involved, whereas if the lesion involved more the

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6. van Rijnberk, G.: Tentativi di localizzazioni funzionali nel cervelletto (il lobulo semplice), Arch. di fisiol. **2**:18, 1905; Weiter Beiträge zum Lokalisationsproblem in Kleinhirn, Ergebn. d. Physiol. **12**:533, 1912.

7. Rothmann, M.: Demonstration zur Physiologie des Kleinhirnwurmis, Neurol. Centralbl. **30**:168, 1911; Zur Kleinhirn Lokalisation, Berl. klin. Wchnschr. **1**:336, 1913; Die Funktion des Mittellappens des Kleinhirns, Neurol. Centralbl. **32**:987, 1913.

8. Thomas, A., and Durupt: Localisations cérébelleuses, Paris, Vigot Frères, 1914.

9. Pagano, G.: Studi sulla funzione del cervelletto, Riv. di pat. nerv. **7**:18, 1905.

10. Luna: Ricerche fatte nel Laboratorio di anatomia normale di Roma, Rome, Università di Roma, 1906, vol. 12, p. 199.

11. Rossi, G.: Ricerche sulla eccitabilità della corteccia cerebrale in cani sottoposti ad emiestirpazione cerebellare, Arch. di fisiol. **10**:251, 1912.

12. Simonelli, G.: Localizzazioni cerebellari corticali, Arch. di fisiol. **20**:405, 1922; Sulle localizzazioni cerebellari corticali e sul loro significato in rapporto alla funzione del cervelletto, ibid. **19**:391, 1914; Contributo alla conoscenza delle localizzazioni cerebellari, Riv. di pat. nerv. **19**:347, 1914.

13. Ingvar, Sven: Zur Phylo- und Ontogenese des Kleinhirns, Folia neurobiol. **11**:205, 1920.

medial portion of the same lobuli, the involvement was more pronounced in the movement of abduction.

Sven Ingvar, experimenting on rabbits, expressed the opinion that in the anterior portion of the lobus anterior there is a center for walking backward and in the lobus posterior a center for walking forward.

Hoshino believed that in the lobus simplex (vermal portion) there is a center for the regulation of movement of the eyes.

Goldstein,[14] in a recent monograph on the cerebellum, expressed the thought that there is a certain amount of localization in this organ and believed that in crus I there is a center especially related to the anterior extremity, while in crus II the center is related to the posterior extremity. It is also his opinion that the muscles of the head are regulated by the lobus anterior and the muscles of the neck by the lobus simplex, and that the lobus posterior has a correlation with the musculature of the trunk and with the roots of the legs, especially for stretching the vertebral column and for falling backward.

At the last meeting of the American Neurological Association (1930), Mussen [15] reported data that he had collected from the removal of various areas of the cerebellum and that seem to confirm his previous results on cerebellar localization obtained by means of electric stimulation.

This author localized in the lobulus centralis of the vermis and in part of the culmen the centers related to the muscles of the neck. In the lateral lobe (lobus ansiformis) Mussen seems to have located a center for the control of the shoulder, while in the lobulus paramedianus there exists a center dealing with the fore legs. In the paraflocculus this author localized a center for the movement of the trunk, limbs, and tail.

In contrast with the authors who believe in a very specialized localization, others, among them Riley [16] and Tilney,[17] are much more of the opinion that the cerebellum must be considered an organ that regulates as a whole the synergic movement of the body and extremities; they based their assertion on studies of comparative anatomy in which

14. Goldstein, K., Das Kleinhirn, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, V. A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1927, vol. 10.

15. Mussen, A.: The Cerebellum: A New Classification of the Lobes Based on Their Reactions to Stimulation, Arch. Neurol. & Psychiat. **23**:411 (March) 1930; The Cerebellum: A Preliminary Report on the Symptoms Resulting from the Localized Destruction of Individual Lobes, American Neurological Association, Fifty-Sixth Annual Meeting, 1930.

16. Riley, H. A.: The Lobules of the Mammalian Cerebellum and Cerebellar Nomenclature, Arch. Neurol. & Psychiat. **24**:227 (Aug.) 1930.

17. Tilney, F., and Riley, H. A.: The Form and Function of the Central Nervous System, New York, Paul B. Hoeber, Inc., 1921.

no correlation has been found between specialized movement of various areas of the body and the various areas of the cerebellum.

Clinical evidences have also been called in support of the existence of cerebellar localization. In 1914, Mills and Weisenburg [18] advanced certain views on the functional localization of the cerebellum. They placed the function of coordination of the limbs in the lateral lobes, for the upper limbs in the superior surface, for the lower limbs in the inferior surface, the centers for movement of bilaterally active functions in the vermis, those for the head, eyes, tongue, larynx and pharynx in the upper superior surface of the vermis and the truncal movements in the remaining part, both superiorly and inferiorly. Weisenburg and Mill's scheme is the one that takes best into consideration the movement of the trunk, emphasizing the importance of the posterior part of the vermis for the control of the pelvic girdle and lower part of the trunk.

Gordon Holmes,[19] in 1918, as a result of a wide clinical experience with cerebellar lesions due to gunshot wounds reached the following conclusions: 1. Each lateral lobe of the cerebellum influences only the motor functions of the same side of the body. 2. When the vermis is injured or both lateral lobes are involved, articulation and the postures and movements of the head and trunk are more affected than when one lateral lobe only is damaged. 3. No local lesion affects only one limb or a portion of a limb. It may therefore be that the vermis is chiefly concerned in the motor functions of the head and trunk, though these suffer equally when both of the lateral lobes are involved, but, in his opinion, there can be no question of separate representation in the cerebellar cortex of the individual limbs.

CONCLUSIONS

The conclusions to be drawn from our preliminary work on the problem of cerebellar localization may be summarized in the following data (fig. 19):

1. The lateral lobes of the cerebellum control the synergy as a whole of the muscles of the homolateral side of the body.

2. The lobulus paramedianus seems also to possess synergic control of both fore and hind legs of the same side, the area controlling the fore leg being, in some animals, located frontally to the area controlling the hind one.

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18. Mills, C. K., and Weisenburg, T. H.: Cerebellar Symptoms and Cerebellar Localization, J. A. M. A. **63**:1813 (Nov. 21) 1914.

19. Holmes, G.: The Symptoms of Acute Cerebellar Injuries from Gunshot Wounds, Brain **40**:461, 1917.

3. Crus I of the lobus ansiformis controls the synergia of both front and hind legs with a predominance over the front one.

Altogether, then, although in the lateral lobe a tendency is observed for the predominance of crus I in the control of the fore leg, there seems to be no definite localized area for either one of the extremities, the removal of any area resulting in an impairment of both front and hind legs.

4. When, to the removal of crus I and crus II, the removal of the paraflocculus is added, asynergia of the tail is a more manifest symptom.

5. The lobus simplex does not seem to possess any specific synergic center either for the neck or for the trunk.

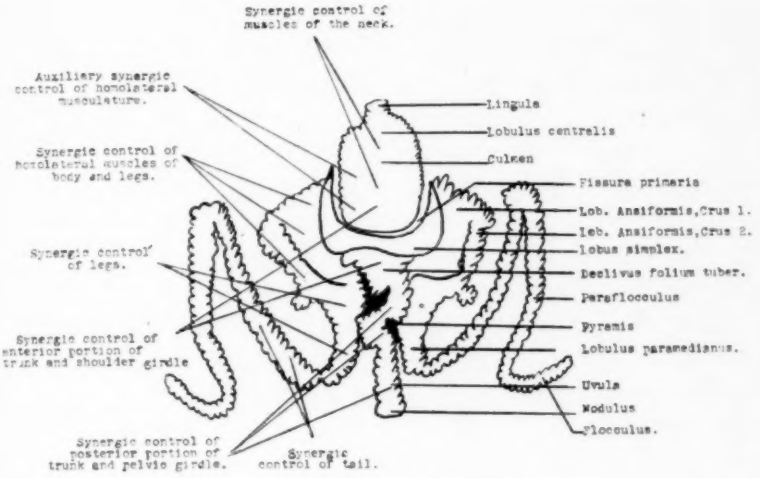

Fig. 19.—Scheme of cerebellar localization resulting from experimental removal of fragmentary portions of the cerebellum.

6. The lateral portion of the anterior lobe, the lobus lunatus anterior (lobulus IV lateralis), seems to participate also in the synergic control of the muscles of the homolateral legs.

7. In the vermis also there seem to be no definite localized centers. This formation seems to control as a whole the musculature of the head, neck and trunk.

8. In the vermal portion of the anterior lobe there is a controlling mechanism for the synergia of the neck located mainly in the culmen (lobulus IV vermalis). The removal of this area results in asynergia of the muscles of the neck without any effect over the rest of the trunk.

9. The remaining part of the vermis is concerned with the regulation of the trunk musculature as a whole, although the clivus, folium,

tuber and part of the pyramis (C^2 presulcal and postsulcal—C^1 vermalis) are dominantly concerned with the synergia of the shoulder girdle and of the muscles of the anterior portion of the trunk. The pyramis, the uvula and the nodulus (C^1 vermalis, lobus B and lobus A) are more concerned with the regulation of the muscles of the pelvic girdle and with the muscles of the posterior portion of the trunk.

10. The removal of the whole vermis is followed by considerable dysmetria of the trunk, especially pronounced over its posterior portion.

11. When to removal of the vermis removal of the lateral lobes is added, as is the case in total decerebellation, the clinical result is a pronounced dysmetria of the trunk, to which dysmetria of the legs is added. The combination results in a clinical picture in which compensation is extremely slow to appear. The loss of coordination following total extirpation of the cerebellum, in the cat at least, is most marked in the posterior half of the body.

OPTIC NYSTAGMUS

IV. PSYCHOLOGIC CONDITIONS *

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Before clinical exploitation of optic nystagmus can be safely attempted, the most complete possible understanding of the normal physiology of this phenomenon must be gained. Early in our work in studying the records of normal subjects the influence of certain psychologic conditions on the nystagmographic pattern was appreciated. In a series of moving visual stimuli of varying complexity, those of little interest commonly evoked only a momentary glance while those of special interest were pursued longer, sometimes to the physiologic limit. Here was an indication that the amplitude of the slow phase might be modified by the psychologic factor called interest. In like manner the amplitude of the quick phase seemed to be influenced by the factor of relative interest engendered by different oncoming objects competing for renewed fixation. In addition, the more complex the object of regard, the greater the number of short quick phases or refixations occurring within the main pursuit.

The opportunity later arose to investigate the influence on optic nystagmus in the normal subject of other psychologic factors, including expectation, habit and learning. A logical method of approach to a problem of this type would be a carefully controlled configuration of the moving field of regard, with experimental variations in the rhythm of succession of the visual objects. Study of the influence of the blind spot also seemed a hopeful procedure. In the experiments that are reported in this paper the different procedures adopted may be regarded as variations in the adequacy of the oncoming visual stimulus.

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* Submitted for publication, Oct. 28, 1930.

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TECHNIC

In previous communications a method for obtaining nystagmograms has been described.[1] It is sufficient to repeat that a mirror recorder is used, in which a concave mirror, pressed against the closed lid of one eye tangential to the underlying corneal surface, reflects a recording beam of light to moving photographic paper.

A special field of view was constructed to carry the visual stimuli, instead of the wire mesh cylinder described in our previous experiments; this was necessitated by the need for a homogeneous background. It consisted of a belt of black

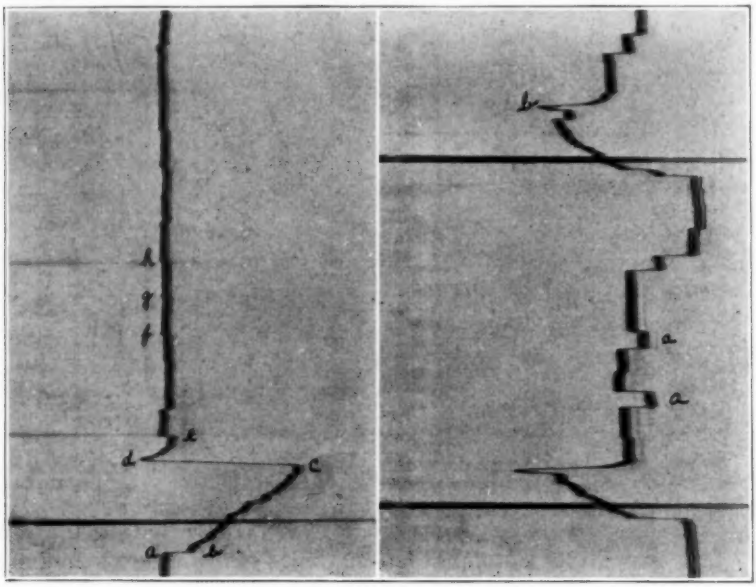

Chart 1 Chart 2

Chart 1.—Subject W.; right eye seeing, left eye recording. Single object passed through the field in a temporal to nasalward direction. Beginning pursuit is characterized by a corrective rapid movement in the direction of pursuit to obtain adequate fixation, *a-b*. True pursuit follows, *b-c*. After the quick phase, *c-d*, there is a brief after-pursuit movement without adequate stimulus which ends in still fixation, *e*. Pulse excursions may be noted at *f*, *g* and *h*.

Chart 2.—Subject B.; right eye seeing, left eye recording. Single object passed through the field in a nasal to temporalward direction. The record is similar to that in chart 1. At *a* are seen drifting still fixations, interrupted by a series of quick searching movements. The second pursuit phase shows two major corrections and is then slightly curved and irregular. This is probably due to an inadequacy in pursuit which is corrected by a rapid movement at *b*, just as the image leaves the field.

1. Dodge, R., and Fox, J. C.: Optic Nystagmus: I. Technical Introduction with Observations in a Case with Central Scotoma in the Right Eye and External Rectus Palsy in the Left Eye, Arch. Neurol. & Psychiat. **20**:812 (Oct.) 1928. Fox, J. C., and Dodge, R.: II. Variations in Nystagmographic Records of Eye Movement, ibid. **22**:55 (July) 1929. Dodge, R.; Travis, R. C., and Fox, J. C.: III. Characteristics of the Slow Phase, ibid. **24**:21 (July) 1930.

velvet 60 inches (152 cm.) in length and 17½ inches (43.8 cm.) in height, which was held in place vertically and which was given a horizontal motion by two cylindrical drums, 1¼ inches (3.1 cm.) in diameter. Springs attached to one of the drums held the belt smooth. Motive power from a direct current electric motor was transmitted by a cord belt to one of the drums. The speed of rotation was regulated by a rheostat. A velocity of 6 inches (15.24 cm.) per second was commonly used and found satisfactory. Under the conditions of our experiments this gave an angular velocity of 16.5 degrees per second. Direction of rotation was changed by twisting the driving belt. In each experiment the angular velocity was kept constant for both directions. The field presented a vertical, flat black wall which completely filled the subject's area of view as far as was permitted by the aperture in the screen before his seeing eye. The belt was placed from 22 to 24 inches (55 to 60 cm.) distant from the eye, thus limiting the moving field of view to an area which was 45 degrees horizontally by 15 degrees vertically.

The objects used for stimuli were the heads of common pins which were bent over at right angles to the shafts and stuck into the velvet. Subdued light was projected to the field perpendicularly, and the heads appeared as bright points of light on the homogeneous black background, making small isolated stimuli of equal intensity. The pins could be moved at will and placed in any predetermined arrangement. When removed, they left no marks on the velvet. The presence of small particles of dust on the velvet was inevitable, but with the low illumination employed they were invisible to the subject and, as our control records show, evoked no nystagmus. It was found that pins spaced at 5½ inches (13.9 cm.) subtended an angle of approximately 15 degrees on the visual field. At this distance, with the subject in place and with the image of one pin on the fovea, the neighboring pin on the temporal side commonly fell in the blind spot and could not be seen. However, the position of the blind spot on the field was always tested empirically for each subject by using a small white object on a black thread. The distance of the field was then so adjusted that the image of the unseen pin was as near the center of the blind spot as possible. Under experimental conditions the invisible pin occupied a point just above the center of a scotoma approximately 1½ inches (3.7 cm.) in diameter.

Each subject was instructed to follow every pin as it crossed his field of view. No other direction was given. The length of pursuit was not specified, but was left to the subject's convenience. As each pin crossed the central point in the field of regard, a horizontal marking line was recorded on the paper. This signal was produced by a small flashing light in front of the exposure slit in the camera for which contact was made through metal clips placed directly beneath each object on the belt.

Various patterns of arrangement of the pins were tried during the course of the experiments. In the records chosen for reproduction in this report the following arrangements were employed: In charts 1 and 2 a single object was placed in the field, to control the presence of casual stimuli which might evoke pursuit.

In charts 3 to 8, the objects were placed 5½ inches apart in regular sequence, arranged so that each oncoming object fell within the blind spot when the field was moving in a temporal to nasalward direction (charts 3, 5 and 7). The purpose of this arrangement was to study the nystagmographic patterns when the oncoming stimulus was invisible from the point of fixation.

In charts 9 to 13, the objects were arranged in various experimental sequences as follows: In charts 9 and 10 there were three pins, 5½ inches apart, a blank space of 11 inches (27.9 cm.) (one pin missing); two pins 5½ inches apart, and a blank space of 11 inches (one pin missing). The sequence was then repeated. In charts

11 and 12 there were two pins 5½ inches apart, a blank space of 16½ inches (41.8 cm.) (two pins missing); two pins 5½ inches apart and a blank space of 22 inches (55.5 cm.) (three pins missing). The sequence was then repeated. These two patterns offered the most satisfactory experimental situation for studying the effects of expectation, habit and learning. In chart 13 the objects were arranged in such a way that a regular sequence of five objects, 5½ inches (43.7 cm.) apart, was followed by a blank space of 32½ inches (82.4 cm.) (six pins missing). This arrangement afforded excellent opportunity for the demonstration of "after-nystagmus."

All records were made by conjugate movements of a closed eye. They read from below upward. The actual direction of movement of the eye is reversed, i. e., movement of the line of regard to the subject's right appears as an excursion to the left. The speed of movement of the eye is shown by the obliquity of the excursions. Still fixation is shown by a vertical line. The greater speed of the rapid phase is shown by relatively finer record lines. Horizontal lines extending across the record indicate the point where the object reaches the center of the field of view. Charts 1 to 8 represent a time interval of eight seconds; the longer durations of charts 9 to 13 is noted under each record. In every instance in which the direction of movement of the field is temporal to nasalward, and in which the influence of the blind spot might be in evidence, the record is marked with a star. All of the records have been reduced for reproduction.

CONTROL OF VISUAL STIMULI

Charts 1 and 2 show that there were no casual stimuli from the moving belt. The velvet presented a perfectly homogeneous background, as evidenced by the complete absence of movements of pursuit, except those evoked by the pin objects.

It is interesting to note that in chart 1 after the quick phase following pursuit, the eye remains in still fixation at the temporal edge of the field, awaiting the anticipated oncoming stimulus. In chart 2, it returns first to the approximate center of the field and then wanders over to the position of the anticipated stimulus. This difference illustrates the individual variations of ocular reaction to a given experimental situation.

INFLUENCE OF THE BLIND SPOT

Adequate Response.—Records of such adequate nystagmus uncomplicated by refixations of any type, as shown in charts 3 and 4, were obtained from only one other subject. The conditions under which these records were made were most carefully controlled. Between the two records the mirror was untouched; only the direction of the moving field was changed. In this subject there is no demonstrable difference in pursuit in either direction, despite the fact that in chart 3 each oncoming stimulus fell within the blind spot. There is no appreciable difference in the adequacy of response as shown in a comparison of charts 3 and 4. Such records are understandable only on the supposition of an anticipatory elaboration of ocular response to unseen but expected stimuli.

Refixation Movements at the Beginning of Pursuit.—Charts 5 and 6 represent a type of reaction that is frequently observed in optic nystagmographic records. The beginning of each phase of pursuit is interrupted by a brief refixation in the opposite direction, with subsequent adequate pursuit. There is little doubt that these refixations occur as a result of an imperfect fixation of the oncoming stimulus. The time that elapses during the brief phase of refixation averages approximately 0.2 seconds, which corresponds to the reaction time of the normal eye. From a casual inspection, in a comparison of charts 5 and 6, it is evident that there are more of these refixations when the

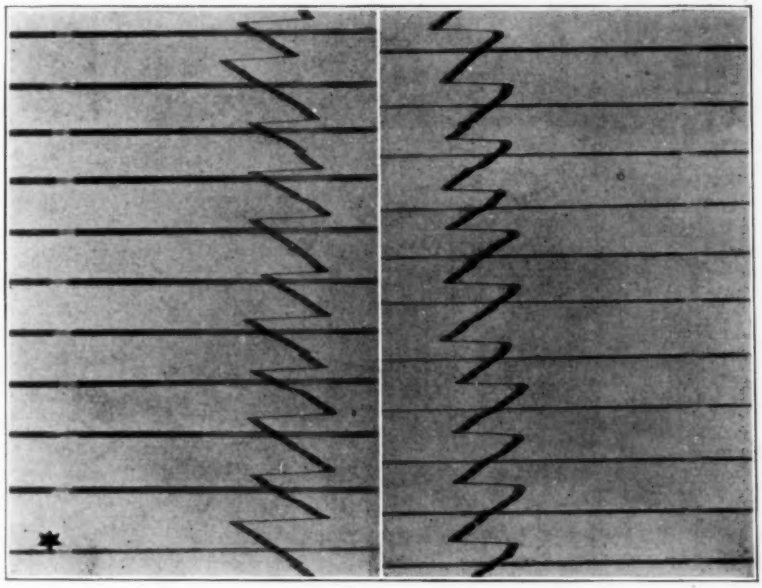

Chart 3 Chart 4

Chart 3.—Subject W.; left eye seeing, right eye recording. Objects were 5½ inches apart in regular sequence, moving in a temporal to nasalward direction. Each new object fell within the blind spot. The record shows normal optic nystagmus.

Chart 4.—Subject W.; left eye seeing, right eye recording. Objects were arranged as in chart 3, but moved in a nasal to temporalward direction. The nystagmographic pattern is regular as in chart 3.

oncoming image is within the blind spot (chart 5) than when the visual stimuli are moving in the opposite direction and the oncoming stimulus is relatively clear (chart 6). Imperfect and corrected fixations of peripheral stimuli are common in all records of the movements of the eye, even with motionless fields. An unseen but expected object the image of which falls within the blind spot represents the lowest degree of clearness, but no peripheral object is really clear.

Nine subjects, whose records revealed relatively large numbers of these refixations, were chosen for quantitative comparison of the percentage of refixations in the two directions of movement of the field. The influence of the blind spot in the production of these is shown by an average of 62 per cent as compared with 38 per cent when the visual stimuli were moving in the opposite direction.

Refixation Movements in the Direction of Pursuit.—Charts 7 and 8 show quick corrective movements in the direction of pursuit. These movements appear much less frequently in the records than the other type of refixations previously discussed. Comparison of charts 7 and

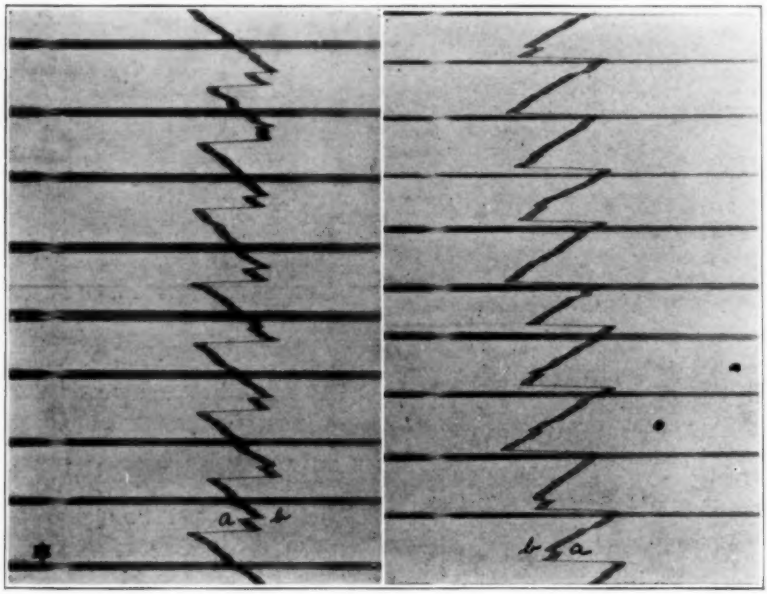

Chart 5 Chart 6

Chart 5.—Subject B.; left eye seeing, right eye recording. Objects were 5½ inches apart in regular sequence, moving in a temporal to nasalward direction. Refixation movements such as *a-b*, appear at the beginning of each phase of pursuit, when the object is in the blind spot.

Chart 6.—Subject B., left eye seeing, right eye recording. Objects were arranged as in chart 5, but moved in a nasal to a temporalward direction. Refixation movements such as *a-b*, occur only irregularly at the beginning of some pursuit phases.

8 reveal that they are definitely more common when the objects are emerging from the blind spot (chart 7) than when the visual stimuli are moving in the opposite direction (chart 8). This picture also represents an incorrect estimate of the position of the oncoming stimulus, but instead of being an underestimate, it is an overestimate.

Chart 14 illustrates diagrammatically the influence of the blind spot on optic nystagmus. At the end of a pursuit such as at *a*, the oncoming

stimulus is unseen because it lies within the blind spot scotoma. The quick phase *a-b* must be determined by other factors, such as habit, anticipation or the fundamental tendency for the eye to return to its primary central position. But at *b* the new object of pursuit is visible, and, after a period of about 0.2 seconds which represents the reaction time of the eye, a corrective movement *c-d* occurs and then true pursuit *d-e* follows. The hypothetical point is represented by *b'*, at which a normal pursuit *b'-e* would have begun if the peripheral retinal stimulation caused by the oncoming object had been adequate at the fixation point *a*.

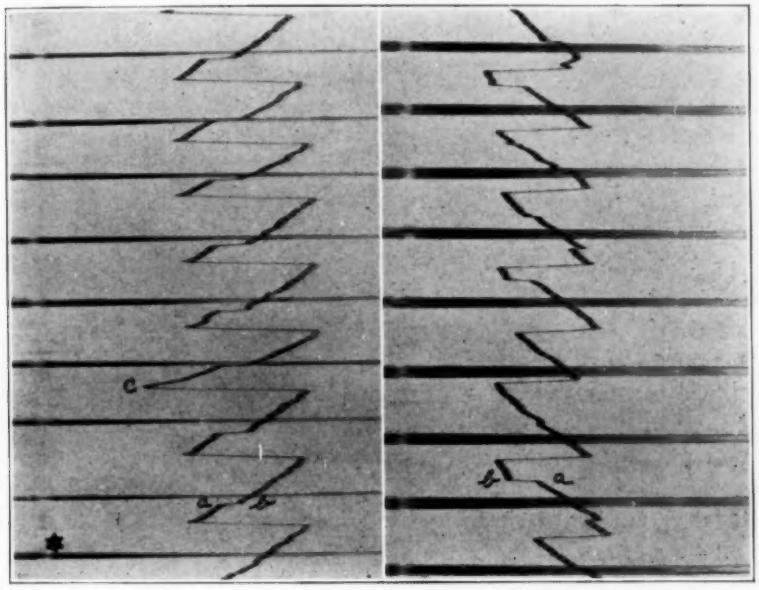

Chart 7 Chart 8

Chart 7.—Subject W., right eye seeing, left eye recording. Objects were 5½ inches apart in regular sequence, moving in a temporal to nasalward direction. Quick corrective movements, such as *a-b* consistently appear in the direction of pursuit. At *c* there appears to be a pronounced over-shooting of the quick phase.

Chart 8.—Subject W.; right eye seeing, left eye recording. Objects were arranged as in chart 9 but moved in a nasal to temporalward direction. The length corrective movements occasionally occur in the direction of pursuit, such as *a-b*.

The presence of a similar phenomenon when the objects were not emerging from the blind spot as in chart 8 can be understood by the fact that peripheral stimulation of the retina is always less adequate for optic nystagmus than foveal stimulation. In other words, stimuli falling on the periphery of the retina are also relatively inadequate, although as a rule they are more effective than those falling within the blind spot.

COMPARISON OF THE RIGHT AND LEFT EYES

Charts 5 and 6 were taken with the left eye seeing and the right eye recording; charts 7 and 8 are from the right eye seeing and the left eye recording. In subjects B and W, a complete series was obtained from each eye. So far as could be determined, the side of the seeing eye, whether right or left, made no difference in the nystagmographic pattern. A directional difference which might be determined by visual habits, such as in reading from left to right, was not found.

PSYCHOLOGIC CONDITIONS—EXPECTATION, HABIT AND LEARNING

Charts 9 and 10 reveal a pursuit pattern complicated by "after-nystagmus." In chart 9, this phenomenon is particularly well illustrated. The "after-nystagmus" was consistently present in all such analogous records of subject A. It is obvious that when the objects are emerging from the blind spot (chart 9), "after-nystagmus" is pronounced, and there are occasional corrective saccadic movements in the direction of pursuit. In chart 10 on the contrary, with objects passing in the opposite direction, pursuit, in the main, is smooth and unbroken and during the intervals when there is no visual stimulation, there is still-fixation instead of "after-nystagmus."

In chart 9, during the intervals without visual stimuli, the "after-nystagmus" is so pronounced that it seems probable that it would have continued for a somewhat longer period of time, had it not been interrupted by a normal pursuit. If the beginning of true pursuit fell directly in with the slow phase of one of the "after-nystagmus" movements, the two would be indistinguishable. The interplay between "after-nystagmus" and true objectively conditioned nystagmus helps to explain not only the occasional normal nystagmus that occurs when objects come from the blind spot, but also the sharp onset of the slow movement after every quick phase without measurable reaction time. Both processes indicate the preelaboration of the phase of pursuit of optic nystagmus by some sort of an integrative process rather than by a chain reflex.

Charts 11 and 12 show the nystagmographic pattern in response to an even more irregular sequence than the arrangement in charts 9 and 10. This particular arrangement of the objects presents greater difficulty for effective preelaboration by the subject since the time intervals between sets of stimuli are unequal. "After-nystagmus" is as well sustained in chart 12 as in chart 11. It should be noted that not once, when the oncoming object was within the blind spot, did there appear two single pursuits for any set of two objects. This is definite evidence that so long as it fell within the blind spot the second

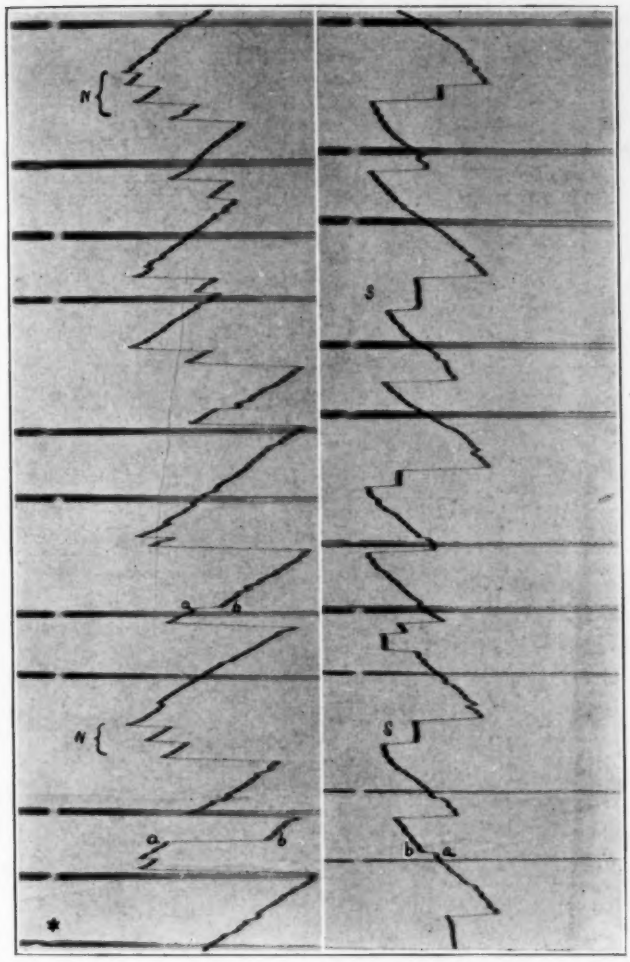

Chart 9 Chart 10

Chart 9.—Subject A.; right eye seeing, left eye recording. Objects were in irregular sequence with three objects missing moving in a temporal to nasalward direction. The next oncoming object of each sequence fell within the blind spot. The length of the record is sixteen seconds. Pursuit is fairly adequate, but there are occasional quick corrective movements within the second phase of pursuit of each series, such as *a-b*. During the brief intervals on the record, corresponding to the blank spaces on the field there is obvious "after-nystagmus," such as at *N*.

Chart 10.—Subject A.; right eye seeing, left eye recording. Objects were arranged as in chart 9 but moved in a nasal to temporalward direction. The length of the record is sixteen seconds. Pursuit is adequate as in chart 9. There are rare quick corrective movements in the direction of pursuit, such as *a-b*. Instead of "after-nystagmus," as in chart 9, there is still fixation during the intervals on the record corresponding to the blank spaces on the field, such as at *S*.

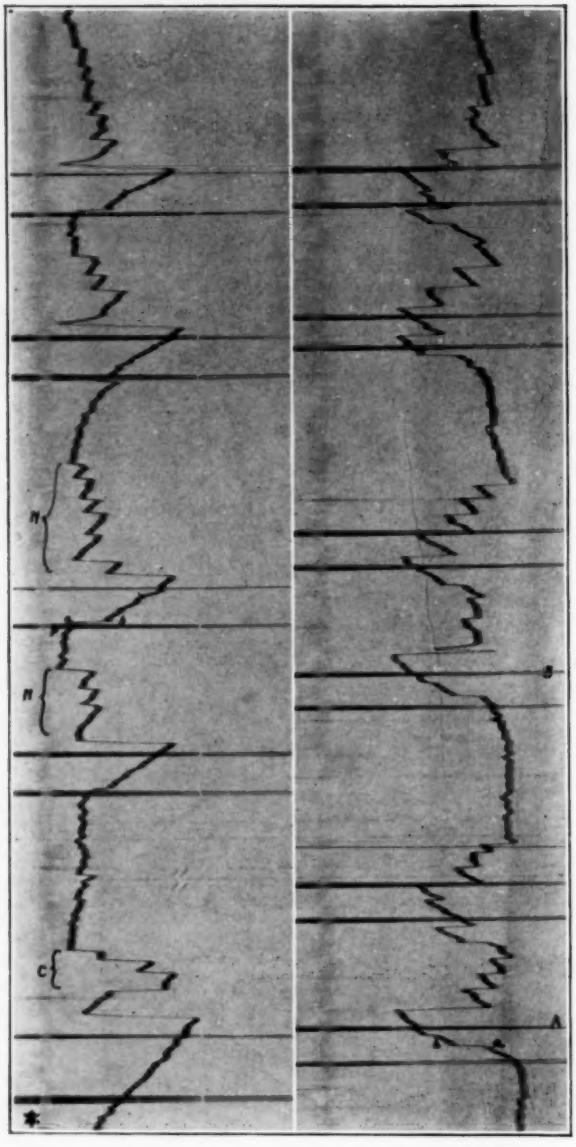

Chart 11 Chart 12

Chart 11.—Subject C., right eye seeing, left eye recording. Objects were arranged in irregular sequence with five objects missing and moved in a temporal to nasalward direction. The length of the record is twenty-one seconds. Only the first object in each pair is pursued. Pursit is usually initiated by a quick corrective movement in the direction of pursuit, such as *a-b*. At *c* there is an interval of "searching" or "groping." "After-nystagmus," such as in periods marked *N*, gradually fades to still fixation before the advent of the next visual stimulus and beginning pursuit such as at *P*.

Chart 12.—Subject C.; right eye seeing, left eye recording. Objects were arranged as in chart 11, but moved in a nasal to temporalward direction. The length of the record is twenty-one seconds. In two instances, marked *A* and *B*, only the first object of a series of two is pursued. The response apparently becomes more adequate near the end of the record. There is consistently a quick corrective movement in the direction of pursuit near the onset of the slow phase, such as *a-b*. "After-nystagmus" is present but is not so well sustained as in chart 13.

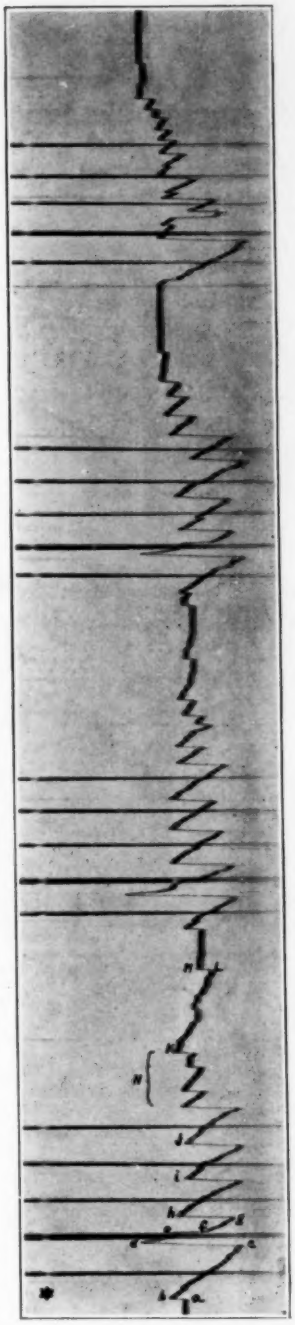

Chart 13.—Subject W.; right eye seeing, left eye recording. Five objects were arranged 5½ inches apart with intervening blanks of 32½ inches between series. The length of the record is twenty-seven seconds. The record shows optic nystagmus in response to three series of five visual stimuli each, and is remarkably consistent throughout. On the appearance of the object a quick movement *a-b* takes place. The pursuit movement, *b-c*, is unusually smooth. The next quick phase beginning at *c* overshoots to *d*. The following pursuit movement *d-g* is interrupted by a corrective after-pursuit glide, *e-f*. The next three oncoming objects are correctly estimated as evidenced by the relatively smooth pursuits beginning at *h*, *i* and *j*. The "after-nystagmus" during the interval marked *N* gradually decreases in velocity and amplitude to the point marked *k* at which a drift carries the eye nasalward to the point marked *L*. This is terminated by a movement temporalward, *L-M*. Still fixation follows until the advent of the next visual stimulus.

pin in the series did not evoke pursuit. In chart 12, learning probably accounts for the relatively more adequate pursuit toward the end of the record.

Chart 13 represents further evidence of habit and learning. Consistently at the end of the rapid phase following pursuit of the first object of each series there is overshooting with a corrective after-pursuit glide in the next pursuit. On the other hand, the relative smoothness of the remaining movements of pursuit indicate a correct estimation of the position of the oncoming objects, although they fall within the blind spot throughout the sequence. This would appear to be a consequence of some process of learning.

RELATION BETWEEN ADEQUACY OF STIMULUS AND "AFTER-NYSTAGMUS"

The records of ten subjects were examined with particular reference to the amplitude and duration of "after-nystagmus," comparing the response in the two directions of movement of the field to determine

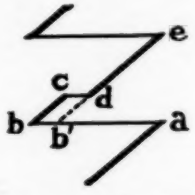

Chart 14.—Diagrammatic illustration of the influence of the blind spot on optic nystagmus.

whether the "after-nystagmus" might be influenced by inadequacy of the stimulus. Three different patterns of irregular stimulus frequency were employed, as shown in charts 9, 10, 11, 12 and 13. The records of five subjects show the "after-nystagmus" to be more marked when the oncoming object is emerging from the blind spot, moving in a temporalward to nasalward direction. On the other hand, the records of five other subjects reveal no significant difference in the "after-nystagmus" depending on the movement of the field. While not capable of statistical statement, the experimental evidence thus obtained indicates that, in some subjects at least, the neuromuscular excitation is more sustained following a succession of objects emerging from the blind spot. In other words, the motor habits of the eye seem to be longer lived under the influence of inadequate stimuli.

To recapitulate, charts 9, 10, 11, 12 and 13 reveal various factors of the nystagmographic pattern conditioned by expectancy, habit and learning. Such determinants must be taken into consideration in all attempts to understand the mechanism underlying optic nystagmus or to explore its application to clinical problems.

SUMMARY

The influence of psychologic determinants on optic nystagmus has been studied in twelve normal subjects by means of nystagmograms, taken with a mirror recorder of movements of the eye.

For this purpose a special moving field of black velvet was constructed to present an undifferentiated black background without configuration, which, on experimental test, evoked no nystagmus.

The adequacy of the oncoming visual stimulus was varied experimentally by spacing the visual objects in such a way that when the image of one was at the fovea, the image of the succeeding object on the temporal side fell within the blind spot and was unseen.

Under this experimental situation the disturbance in visual fixation at the beginning of pursuit was shown by an increase in the number of quick refixations which occurred just prior to the beginning of true pursuit and also within the pursuit phase in the direction of pursuit.

Carefully controlled variation in the rhythm of succession of the visual objects, permitted an analysis of the normal nystagmographic pattern and demonstrated the interplay of certain psychologic factors, including expectation, habit and learning.

On the sudden cessation of all visual stimuli, "after-nystagmus" usually follows for a brief interval of time.

The "after-nystagmus" is more striking following a succession of inadequate than after adequate visual stimuli.

When "after-nystagmus" is interrupted by true pursuit in response to a new stimulus, the characteristics of the two reactions are indistinguishable and indicate the preelaboration of the normal pursuit phase of optic nystagmus by some sort of integrative mechanism, including expectation, habit and learning, as well as adequate stimulation.

CEREBRAL ANEURYSM AND CORTICAL HERNIATION *

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AND
A. E. BENNETT, M.D.
OMAHA

Cerebral aneurysms are of greater frequency than is ordinarily considered. The hemorrhage from a ruptured aneurysm is too easily diagnosed as cerebral or meningeal apoplexy, and the origin of the hemorrhage is not verified by necropsy. Cortical herniation into dural sinuses is another condition that occurs rather frequently but is not commonly recognized. It is found in association with increased intracranial pressure. Neither condition often presents localizing diagnostic symptoms, but these may occur and be significant. This report of ten cases of cerebral aneurysm, eight of which were verified by necropsy, is prompted because of the application of internal carotid ligation in two and the discovery of an unusual complication of herniation of the motor cortex into pacchionian bodies in another. Before presentation of the case reports, a few of the significant points pertaining to the two subjects will be discussed briefly.

The subject of cerebral aneurysm has recently been presented in an excellent manner by Sands [1] and by Parker; [2] a further review of the literature is not warranted. The majority of cerebral aneurysms are located on the arterial circle of Willis at the base of the brain. The commonest site is at the place of origin of the posterior communicating artery from the internal carotid, which was found in six cases of our series. The pathogenesis of cerebral aneurysm is not entirely clear, although anatomic factors, septic emboli and arteriosclerosis are given as the three most common causes, in the order named.

Congenital weakness of the cerebral arterial walls is explained on the basis of several anatomic peculiarities. The cerebral arteries at the base of the brain are unprotected by abundant surrounding connective tissue or adventitia. The same is true of smaller vessels that enter the central nuclear masses. Several rather small branches arise from large arterial trunks, particularly the posterior communicating branch from the internal carotid artery. This necessitates that relatively

* Submitted for publication, Nov. 28, 1930.

* From the Neurological Service, University of Nebraska College of Medicine.

1. Sands, J. J.: Aneurysms of the Cerebral Vessels, Arch. Neurol. & Psychiat. **21**:37 (Jan.) 1929.

2. Parker, H. L.: Aneurysms of the Cerebral Vessels, Arch. Neurol. & Psychiat. **16**:728 (Dec.) 1926.

small and unprotected arteries carry the high arterial pressure of the main trunk, without the reduction that occurs in the usual gradual subdivision of an artery to form a capillary bed. Both the posterior communicating and the anterior communicating arteries of the circle of Willis are subject to cross strain or pull by the two larger arteries connected. And finally there is a recognized anatomic weakness of the arterial walls at points of bifurcation, due both to the disarrangement of muscle fibers and to the constant impact of the blood current at the angle of division.

Septic emboli definitely are the cause of a limited number of cerebral aneurysms. These usually occur during the course of a vegetative endocarditis which runs a septic course, to terminate in death within a few weeks. Less well defined are the emboli that come from other infections, particularly pulmonary, without a high degree of sepsis. Some infections of a nonsuppurative type tend to localize in arterial walls or perivascular tissue. It is not often possible to evaluate all of these infectious factors in the analysis of a case of cerebral aneurysm.

Cerebral arteriosclerosis of advanced age rarely gives rise to aneurysm, except occasionally to multiple miliary aneurysms in the smaller branches of the cerebral arteries. Hundreds of these may be found, and are best demonstrated by macerating the brain tissue and washing it away with an active current of water. Some writers [3] have claimed that this type of aneurysm is related to the common cerebral hemorrhage or apoplexy, but the size would seem to be too small to cause much hemorrhage by rupture. The hypertension that usually accompanies cerebral arteriosclerosis is a factor in the rupture. This type of arteriosclerotic miliary aneurysm is distinct from the larger aneurysm that is found on the arteries at the base of the brain. The latter usually is from 0.5 to 1 cm. in diameter, is isolated and occurs with surprising frequency in persons under 50 years of age in whom there is little evidence of cerebral arteriosclerosis.

Syphilis rarely is the cause of cerebral aneurysm. Syphilitic aneurysm characteristically involves the larger arteries, particularly the arch of the aorta, by obliteration of the vasa vasorum and a consequent degeneration of the arterial wall. Few of the cerebral arteries are large enough to be thus affected. In only one of the ten cases here reported did the blood or spinal fluid give a positive Wassermann reaction, a proportion that corresponds to the infrequent incidence of syphilis in other reported cases.

The clinical symptoms of cerebral aneurysm are not often diagnostic before rupture. Localized throbbing pain, with involvement of

3. Sherman, T.: Miliary Aneurysm in Relation to Cerebral Hemorrhage, Edinburgh M. J. **15**:825, 1915.

the basilar nerves and absence of intracranial pressure should make one think of a possible aneurysm in distinction from a tumor of the brain. The sudden basilar subarachnoid hemorrhage, with symptoms of meningeal irritation and acute intracranial pressure, particularly in a person under 50 years of age, with no evidence of blood disease, trauma or acute infection, should lead to a presumptive diagnosis of ruptured cerebral aneurysm.[4]

The frequency of spontaneous meningeal hemorrhage has not attracted as much attention in this country as it has in reports from Europe. Seven additional cases of suspected cerebral aneurysm might be added to our series, but are not included on account of lack of localizing signs or postmortem verification. Three have come under observation during the writing of this paper and illustrate the frequency of the condition. The diagnosis of pachymeningitis haemorrhagica should always be questioned as having a possible source from an aneurysm. This is well illustrated by case 5. The relation of trauma to rupture of a cerebral aneurysm may present problems in interpretation, as in case 8. Subdural instead of subarachnoid hemorrhage, as in case 3, might be confused with chronic subdural hematoma of venous traumatic origin.

The treatment for cerebral aneurysm is limited to palliative measures, for removal of the lesion is impossible. Although spinal drainage seems to be indicated for relief of pressure and removal of blood, it may be dangerous in continuing or initiating hemorrhage; also the cerebellum may herniate into the foramen magnum as in case 1. Judgment should be based on the seriousness of pressure and meningeal symptoms, which necessitate some attempt at relief. Elevation of the head and quiet for at least a month after the rupture are indicated. Ligation of the internal carotid artery should be of benefit in reducing the systolic impulse into the aneurysm, particularly when the aneurysm can be located on the internal carotid or one of its branches by unilateral oculomotor or cerebral symptoms, as in cases 2, 3, 9 and 10. The dangers of carotid ligation must be kept in mind; they are due chiefly to an extending thrombosis which obliterates the cerebral circulation on one side.

Herniation of the cerebral and cerebellar cortex into arachnoid granulations or pacchionian bodies is a well recognized complication of high grade increased intracranial pressure. According to Wolbach,[5] such hernias are of constant occurrence, and failure to report them in necropsy protocols is due to lack of careful examination of the sinuses

4. Leopold, S. S.: Spontaneous Subarachnoid Hemorrhage, M. Clin. North America **13**:869, 1930.

5. Wolbach, S. B.: Multiple Hernias of the Cerebrum and Cerebellum, Due to Intracranial Pressure, Am. J. M. Sc. **19**:153, 1908.

of the dura mater. They occur in a most striking manner in cases of tumor of the brain or internal hydrocephalus, but may occur acutely in cerebral hemorrhage. Enlargement of the pacchionian granulations occurs with age and appears to be influenced by pathologic conditions which produce a low grade increase of cerebrospinal fluid pressure, as in dementia paralytica, chronic nephritis and vascular hypertension. Case 1 presents an unusual enlargement of two pacchionian granulations overlying the motor cortex, with herniation and thrombosis of this cortex.

REPORT OF CASES

CASE 1.—*History.*—A white woman, aged 44, married, entered the Clarkson Hospital in a stuporous condition on Oct. 27, 1928, at 10:45 a. m. Convulsions had suddenly developed at 4 a. m. on that day, and she continued in status epilepticus until relieved by large doses of morphine. She had had severe pain in the right ear and temple shortly preceding the convulsions, but no headache. She had been troubled with asthma for several years and had had a right chronic otitis media fifteen years before.

Examination.—On admission, the patient was conscious but very sluggish; she was perspiring profusely, and appeared dehydrated, exhausted and cyanotic. Neurologic examination, by Dr. George E. Neuhaus, showed the pupils to be slightly dilated and irregular and reacting poorly to light. The optic disk was congested and blurred, but there was no elevation. The remaining cranial nerves were normal. The tendon reflexes were hyperactive; ankle clonus was positive on both sides. The plantar reflexes were normal, thigh flexion slightly positive and the neck rigid. The abdominal reflexes were present; reaction to pain appeared normal, although cooperation was poor. The patient moved with difficulty, owing to exhaustion and a feeling of numbness or stiffness over the entire body. She complained of severe headache and nausea. The rectal temperature was 102 F., the pulse rate 70 and respirations 22; the leukocytes of the blood numbered 24,800.

Diagnosis.—Meningitis with an origin either from a ruptured abscess of the brain or from a subarachnoid hemorrhage.

Course.—A spinal puncture revealed a very bloody fluid, under a pressure of 40 mm. of mercury, with a red cell count of 1,240,000, and leukocytes 1,000 per cubic millimeter. The patient's mental reaction improved following drainage of 15 cc. of spinal fluid, but the other symptoms persisted. Repeated punctures were performed for relief of pressure and drainage of blood, with gradual clearing of the spinal fluid and considerable general improvement. Paralysis of the lower extremities and spasticity of the upper extremities persisted. She was able to move the toes slightly. Reaction to touch, pain and deep sensation appeared normal.

On the fifth day, headache and restlessness increased, and the spinal fluid appeared more bloody. At 2 a. m. that night, she suddenly became aphasic and stuporous, and was unable to move the right arm. The condition gradually became worse, with medullary collapse, and death occurred thirty-six hours later.

Necropsy.—There was a ruptured aneurysm of the left anterior cerebral artery with fresh and old clotted blood in the basal subarachnoid spaces and blood staining over the cerebral hemispheres. The aneurysm measured 5 mm. in diameter. There was slight evidence of cerebral arteriosclerosis, and none at the site of the aneurysm. There were unusually large pacchionian bodies along the superior sagittal sinus, two of which in the region of the coronal suture fitted into large

depressions in the inner table of the skull, measuring 2 cm. in width and from 0.5 to 1 cm. in depth. Removal of the dura from the brain disclosed numerous cerebral herniations into the pacchionian bodies. The cortex under the two largest pacchionian bodies did not adhere, but the separated cortical surface was elevated and dark red, and involved the precentral motor gyri just medial to the arm centers (fig. 1). A section through these areas showed an extension of the cortical thrombosis to a depth of 1 cm. (fig. 2). The left cerebral hemisphere was softer than the right, and there was herniation of the cerebellum into the foramen magnum.

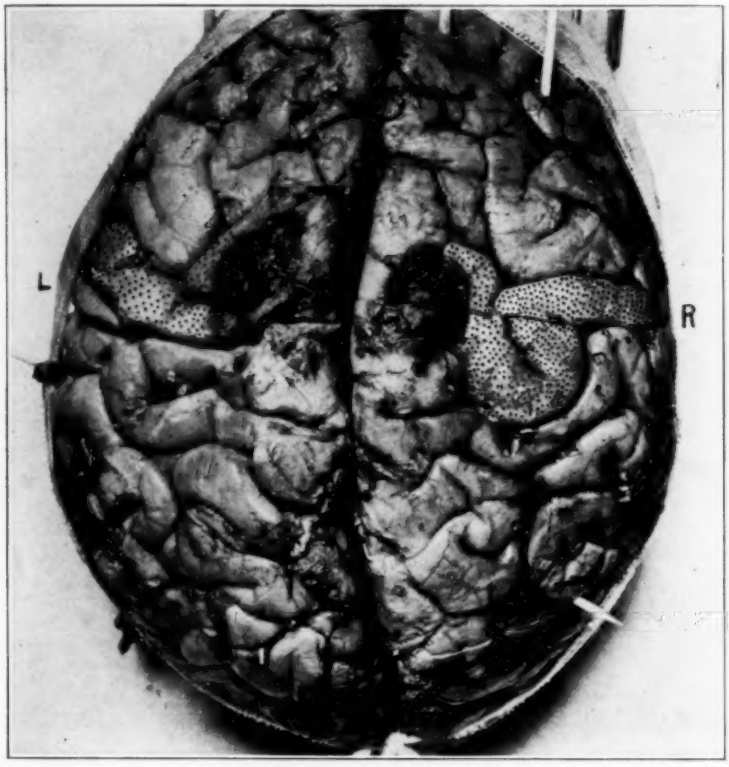

Fig. 1 (case 1).—Thrombosis of the motor cortex from herniation into pacchionian bodies.

Comment.—The etiology of the aneurysm in this case may have been related to the chronic bronchial infection and asthma. The absence of focal cranial nerve symptoms is characteristic of aneurysm of the anterior cerebral arteries. The premonitory pain in the right ear and temple may have indicated the first escape of blood, before the larger subarachnoid hemorrhage and pressure initiated convulsions. The irritant effect of blood in the cerebrospinal fluid is well illustrated by the meningeal symptoms that were present. The persistent motor

paralysis of the lower extremities and the spasticity of the upper extremities were not satisfactorily explained before necropsy, but evidently were due to the herniation and thrombosis of the precentral motor gyri into pacchionian bodies. The treatment by repeated spinal drainage was perhaps unwise, particularly in view of the cerebellar herniation into the foramen magnum found at necropsy. However, the signs of meningeal irritation and intracranial pressure seemed to necessitate relief by this means.

Case 2.—*History.*—A colored woman, aged 29, was brought to the University Hospital in a state of coma on July 27, 1921. The illness dated from July 24, at 4:45 a. m., when she was found unconscious in bed. She had been treated at home for "stomach trouble" during the preceding two weeks.

Examination.—On entrance there were stertorous breathing, a rectal temperature of 100.6 F., a pulse rate of 96 and a respiratory rate of 24; the body was

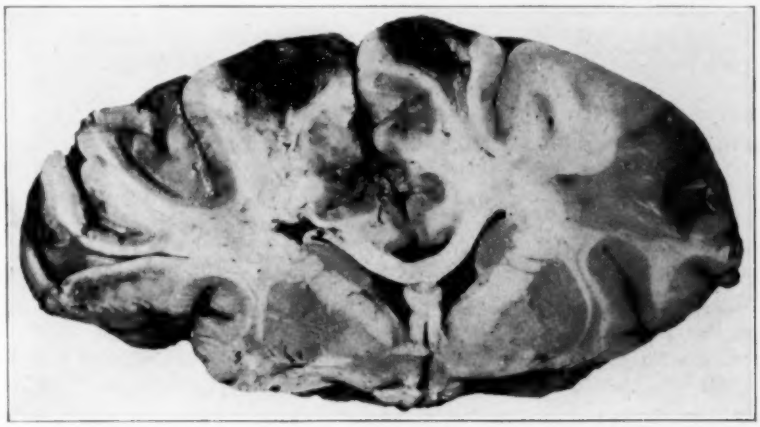

Fig. 2 (case 1).—Thrombosis of the motor cortex from herniation into pacchionian bodies. Cut surface.

relaxed, the eyes staring and the pupils dilated and fixed, the right larger than the left; ptosis of the right lid and left hemiplegia were present. Spinal puncture released blood-stained fluid under moderately increased pressure. The blood leukocytes numbered 15,400; the Wassermann reaction of the blood and spinal fluid was negative.

Course.—The patient remained in a state of coma, with rising temperature, and pulse and respiratory rate, and died on Aug. 1, 1921.

Necropsy.—There was a diffuse red staining of the subarachnoid space, which was more marked over the right cerebrum. A large amount of blood was found in the basal cisternae. There was a firm blood clot beneath the right crus cerebri, which surrounded a ruptured sacculated aneurysm, 1 cm. in diameter, arising from the right internal carotid artery just before its division into the anterior and middle cerebral branches. There was considerable pressure necrosis of the under surface of the right temporal lobe. A similar unruptured aneurysm was found in an identical location on the left internal carotid artery. Careful dissection of all cerebral vessels disclosed no other aneurysms and no arteriosclerosis.

Comment.—This case perhaps falls into the classification of congenital arterial defect, since the two aneurysms found were almost identical in location and size. The preceding history is too brief and incomplete to permit early diagnostic interpretation. The right ptosis and left hemiplegia were caused by pressure of the hemorrhage on the right third nerve and the crus cerebri (Weber's syndrome), and indicated the location of the ruptured aneurysm. Ligation of the right internal carotid artery was indicated.

CASE 3.—*History.*—An obese colored woman, aged 46, was brought to the University Hospital in a stuporous condition on Oct. 28, 1921, moaning, with pain in the neck and left side of the head. There was a history of attacks of dizziness, beginning three or four weeks previously, accompanied by nausea and vomiting. These symptoms gradually increased in severity, with pain in the posterior cervical and left supra-orbital regions.

Examination.—There were sluggish reaction of the pupils to light and in accommodation, partial ptosis of the left lid and slight lateral nystagmus, which was not constant; the left eye did not converge. The optic disks were elevated 2 diopters. There was a slight decrease of motor and sensory reaction on the right side. The biceps, triceps and patellar tendon reflexes were absent; the plantar reaction was normal. The temperature was 98.4 F., the pulse rate 60 and respirations 24. Spinal puncture on October 29 showed blood-tinged fluid under greatly increased pressure, with a red cell count of 22,000 per cubic millimeter. The Wassermann reaction of the blood and spinal fluid was negative; the blood pressure was 145 systolic and 85 diastolic.

Course.—The left ptosis and oculomotor weakness improved, as did the patient's general symptoms, so that she was sitting up in bed and conversing with other patients on November 13. On November 16, she suddenly became comatose, with labored breathing, the left eye fixed, profuse perspiration, a slow and bounding pulse and convulsions, and she died about three hours later with an elevation of temperature, and of pulse and respiratory rate.

Necropsy.—There was an extensive left subdural blood clot, increasing in size toward the under surface of the temporal lobe. There was very little blood in the subarachnoid space. The blood clot extended from the base of the brain upward along the left side of the falx cerebri and into the posterior fossa. The source of the hemorrhage was a small ruptured aneurysm, about 5 mm. in diameter, situated on the left internal carotid artery at the place of emergence of the posterior communicating branch of the circle of Willis. No other aneurysm was found in the cerebral arteries. There were a few yellow intimal plaques in the basilar artery, but none elsewhere.

Comment.—This case presents an unusual subdural extension of the hemorrhage. The aneurysm may have been arteriosclerotic, although the evidence was very slight. The pain in the left side of the head and the left oculomotor paralysis indicated the location of the lesion on the left internal carotid artery. Ligation was indicated, but was not performed.

CASE 4.—*History.*—A white man, a farm laborer, aged 27, was brought to the University Hospital on Nov. 17, 1923, having been found unconscious in a corn-

field shortly after he had gone to work. There was a history of severe headaches since 12 years of age of a migraine type, accompanied by nausea. Two years before, he had had an attack of pain in the head when getting ready for bed; he cried out and sank to the bed, and was unable to speak or see for about an hour, but could hear what was said to him. There was no paralysis and he recovered completely in a few days. Since then he had had frequent and severe headaches, made worse by stooping or working in the hot sun. On November 15, he had a severe headache and remained at home. On the morning of November 17, with no complaint of pain, he had gone to the field to husk corn. A fellow worker found him lying unconscious on the ground by his wagon. He had had influenza in 1918, with a relapse and a stuporous condition for several days; he was sick in bed for two weeks.

Examination.—He was a well nourished man in coma, cyanotic, with stertorous breathing and constant movement and twitching of the muscles of the right side, but with no movement of the left arm or leg. The head was turned to the left and the neck was rigid. The pupils were small and regular and reacted to light, and the eyes were divergent. The retinal vessels were engorged and the margins of the disks blurred, but with no elevation. Spinal puncture released blood-stained fluid under 28 mm. of pressure. The blood pressure was 122 systolic and 76 diastolic; the Wassermann reaction of the blood was negative.

Course.—The patient's condition improved following the spinal puncture; he regained consciousness and began to move the left arm and leg. On November 20, he complained of severe temporal headache and his temperature rose to 103.5 F. A second spinal puncture showed the fluid still bloody and under 17 mm. of pressure. Another attack of severe temporal pain occurred during the night of November 24. The following noon, he began to perspire profusely and became unconscious; a left hemiplegia recurred. Spinal puncture showed a pressure of 30 mm. of mercury, with increased bloodiness of the fluid. He remained comatose; the temperature, and pulse and respiratory rate gradually rose, and he died on Nov. 29, 1928.

Necropsy.—There was a ruptured aneurysm, 5 mm. in diameter, located on the right posterior communicating artery of the circle of Willis, near its origin from the right internal carotid artery (fig. 3). The basilar subarachnoid spaces were filled with blood, which extended over the cortex as blood staining. There was a larger clot about the aneurysm and evidence of pressure on the base of the right cerebral hemisphere. The left hemiplegia located the lesion on the right internal carotid artery, and ligation might have been of benefit.

CASE 5.—*History.*—A white woman, aged 50, a patient of Dr. A. D. Dunn, died on Sept. 1, 1926. The history of illness antedated death about eight years, and the case was reported by Dr. Dunn in 1922 under the title of "Pachymeningitis Hemorrhagica Interna."[6] The first symptoms, eight years previously, were the sudden onset of intense pain in the back of the head and neck, quickly followed by general convulsions and unconsciousness for several hours, with signs of meningeal irritation and slight right facial weakness. Spinal puncture revealed a uniformly bloody fluid. During a period of three weeks, there were four similar attacks, each time with fresh blood in the spinal fluid. She recovered entirely after a convalescence of eight months and there was no recurrence of the trouble during the following four years. She was seen again in June, 1925, with a com-

6. Dunn, A. D.: Pachymeningitis Hemorrhagica Interna, Am. J. M. Sc. **163**:819, 1922.

plaint of weakness and difficulty in walking. She had a positive Romberg sign, nystagmus and ataxia; the systolic blood pressure was 210, and the diastolic 125; the Wassermann reaction of the blood was negative. The Wassermann reaction of the spinal fluid was negative. The weakness gradually increased, and on the night of death there were signs of progressive medullary failure with sudden cyanosis, diaphoresis and death.

Necropsy.—There was an oval aneurysm of the left vertebral artery, about 2.5 cm. in diameter, which markedly compressed the medulla (fig. 4). There was no subarachnoid hemorrhage.

Fig. 3 (case 4).—Ruptured cerebral aneurysm on the right posterior communicating artery of the circle of Willis.

Case 6.—*History.*—A white man, aged 56, a patient of Dr. A. D. Dunn, entered the Clarkson Hospital in coma, with a history of sudden collapse a few hours previously. He died shortly after entrance.

Necropsy.—There was a massive basilar subarachnoid hemorrhage, extending in lesser degree over the convexity of the cerebrum. A ruptured aneurysm of the right vertebral artery was found, measuring from 5 to 10 mm. in diameter. No other aneurysm of the cerebral vessels was found. The Wassermann reaction of the blood was negative.

Case 7.—*History.*—The patient, aged 31, entered the Wise Memorial Hospital on March 21, 1927, on account of severe headache, convulsion and vomiting. The

past history was without significance except for attacks of migraine since childhood. On March 9, a sudden pain developed over the right eye and right temporal region, followed by vomiting which lasted for several hours. The pain had been present intermittently since the onset. On March 17, he fell on the street in a convulsion which was followed by another attack of vomiting.

Examination.—Physical and neurologic studies gave essentially negative results; slight fever was present; the leukocyte count was 14,500; the spinal fluid was under 14 mm. of pressure and uniformly bloody. The Wassermann reaction of the blood was negative.

Course.—Following daily lumbar punctures and saline catharsis the clinical symptoms disappeared, and the spinal fluid became normal. The patient insisted on leaving the hospital prematurely. While in his berth at the station, following

Fig. 4 (case 5).—Aneurysm of the left vertebral artery, causing death by pressure on the medulla.

coitus, he experienced severe pain, became unconscious and died of medullary collapse. A cisternal puncture, performed just prior to death, gave very bloody fluid.

Necropsy.—There was an aneurysm of the middle cerebral artery on the right side, about 7 mm. in diameter. The aneurysm had broken into the temporal lobe, producing a large blood cyst and diffuse blood in the entire subarachnoid space. The pain in the right side of the head had indicated the location of the lesion on the right internal carotid artery, and ligation might have been of benefit.

Case 8.—*History.*—A man, aged 29, had been in normal health, except for known vascular hypertension, until March 28, 1930, when he slipped and fell into a manhole, striking his coccyx. This jarred him considerably and caused him to lie down for about an hour, but work was resumed without apparent ill effects except for lameness and soreness of the back. Three days later, he had a sudden

convulsion followed by severe headache. The headache was intermittent at first but soon became continuous. Spinal puncture revealed a normal, clear fluid but with a markedly increased pressure. The Wassermann reaction of the spinal fluid was negative. The headache was relieved temporarily, but soon recurred.

Neurologic Examination.—On April 11, there was no evidence of focal disease. The reflexes were all diminished; there was a suggestive bilateral Kernig sign and rigidity of the neck. A spinal puncture revealed uniformly bloody fluid under 30 mm. of pressure; the color was light pink. The Wassermann reaction of the blood was negative. The blood pressure was 170 systolic and 110 diastolic.

Course.—The headaches gradually became more severe and continuous, and the meningeal signs more marked. Repeated spinal and cisternal punctures always

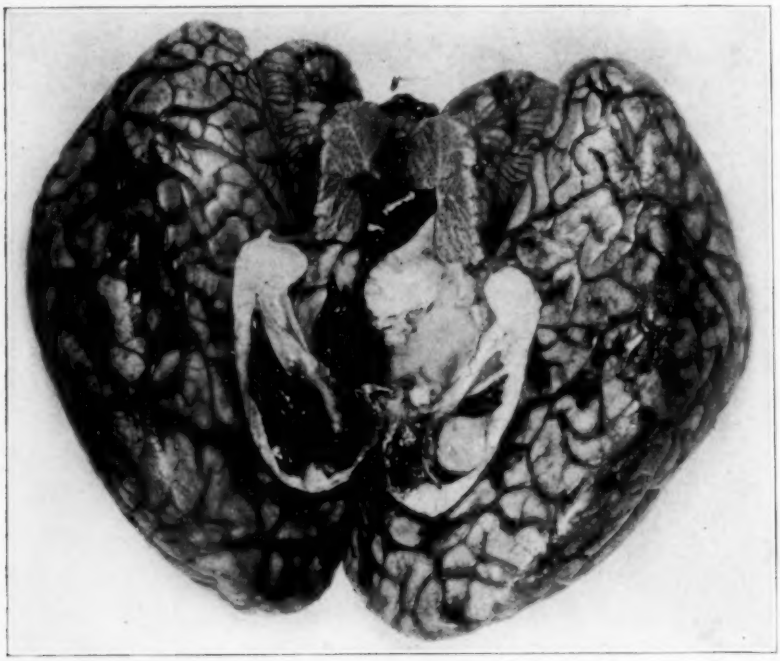

Fig. 5 (case 8).—Aneurysm of the right anterior cerebral artery with rupture into the lateral ventricle and ventricular blood cast.

revealed bloody fluid; for a time it became clearer, then cherry red. At times the patient was delirious. He had moderate fever and leukocytosis. An attempt to localize the bleeding point by cisternal injection of air was made, but the roentgenograms did not show anything abnormal except a failure of the right ventricle to fill completely. The patient suddenly had convulsions on April 23, and died.

Necropsy.—There was an oval aneurysm, about 1 cm. in length, located on the right anterior cerebral artery at the junction with the anterior communicating artery of the circle of Willis. The aneurysm had ruptured upward through the right frontal lobe into the anterior horn of the right lateral ventricle. A blood cast filled this ventricle and also the third and fourth ventricles (fig. 5), with

escape of only a moderate amount of blood into the basal subarachnoid spaces. There was little or no blood anteriorly or over the convexity of the cerebral hemispheres. The left lateral ventricle was dilated, the convolutions were flattened and the right frontal lobe was softened. The cerebral arteries showed several yellow intimal plaques at points of bifurcation, but no evidence of aneurysm elsewhere. The coronary arteries and the aorta showed similar yellow plaques. The heart was large, with hypertrophy of the left ventricle, and a row of small fibrous vegetations was found near the free border of the mitral valves.

Comment.—This case is of interest because of the number of possible etiologic factors involved. The premature arteriosclerosis and hypertension evidently preceded the injury, and may have been responsible for the primary weakening of the cerebral arterial wall. The endocarditis likewise was of some duration, and the possibility of a cerebral embolus from this source must be considered. The aneurysm undoubtedly antedated the injury, but its thinned wall may have been further stretched or partly torn by the jarring of the head, transmitted through the vertebral column. The absence of focal cranial nerve symptoms is typical of involvement of the anterior cerebral artery. The normal spinal fluid obtained at the first puncture is explained by the ventricular hemorrhage with retention of blood in the ventricles an appreciable time before it reached the subarachnoid space.

CASE 9.—*History.*—A woman, aged 52, who was admitted to St. Joseph's Hospital on Oct. 24, 1928, complained of severe suboccipital pain which had begun suddenly two weeks before admission. The pain increased in severity, and within one hour the patient was in coma. She was hospitalized for several days; the pain gradually subsided and except for weakness she felt well. A few days after returning home, the pain recurred over the left eye and temporal region, this time associated with blindness on the left side.

Examination.—Physical examination gave essentially negative results; the blood pressure was 175 systolic and 95 diastolic. Neurologic examination gave negative results, except for loss of pupillary reaction on the left and a sluggish reaction on the right, left optic atrophy with complete amaurosis, and right quadrant temporal hemianopia, the upper field being lost.

Course.—The patient was delirious and stuporous by spells; the temperature ranged from 100 to 102 F., and leukocytosis from 14,650 to 16,200. Rigidity of the neck and a bilateral Kernig sign were present. The sole complaint was severe pain in the left temporal region. A cisternal puncture revealed uniformly bloody spinal fluid, under considerable pressure; drainage relieved the pain somewhat. The Wassermann reaction of the blood was negative.

Spinal drainage was performed several times, but the localized pain on the left side was never completely relieved; at times it required morphine. The opinion was that the patient had a cerebral aneurysm, which had ruptured, producing hemorrhagic meningitis. The ocular changes localized the aneurysm in the region of the optic chiasm on the left side, catching some of the fibers on the right side anterior to the chiasm.

Because of an exacerbation of pain and rising temperature, it was decided to ligate the left internal carotid artery. This was done by Dr. C. A. Roeder on November 7. Except for a short period of tachycardia, dyspnea and precordial

pain, the patient suffered no reaction from the operation. There was a short period of pleurisy and rheumatic pains; then the patient convalesced completely and was discharged on Dec. 11, 1928, entirely relieved from pain in the head. The ocular condition was unchanged.

A recent communication from the patient stated that her general health was much improved and she was able to do all her own work. Total blindness was present in the left eye, but she had good vision in the right. The right side of the neck felt stiff, and there was some pain in this region.

Case 10.—*History.*—A colored woman, aged 43, was admitted to the University Hospital on March 10, 1930, complaining of frontal pain radiating to the occiput, nausea and vomiting, with fainting spells and impaired vision. Two weeks previously she had had severe pain in the frontal region associated with a cold in the head. The nasal obstruction and discharge improved, but the headache became worse and radiated to the occiput. Two nights before admission to the hospital, she felt blood rush to the head and felt very hot. An hour later she became dizzy and fell; she was unconscious for a half hour and on awakening had a severe headache and black spots before the eyes. Attacks of nausea and vomiting followed, each being preceded by a severe pain in the head. The Wassermann reaction of the blood was positive.

Examination.—The patient was obese and lay in bed groaning with pain in the head. She had sudden attacks of vomiting; the neck was rigid and the Kernig sign was positive; the temperature was normal. The pulse rate was slow and irregular; the blood pressure was 170 systolic and 100 diastolic. The pupils were small and reacted slowly to light. Examination of the fundi showed preretinal hemorrhages in both eyes. The margins of the disks were indistinct but not elevated. The nasal sinuses were normal. A cisternal puncture showed a pressure of 35 mm. of mercury and a very bloody fluid.

Course.—The headache and vomiting were relieved considerably by repeated spinal fluid drainage. The pressure and blood discoloration gradually subsided. The patient was discharged on April 16, feeling well except for slight headache, weakness and impaired vision. She had a recurrence of severe headache and vomiting three days later, and was readmitted to the hospital on April 20. Examination then showed ptosis of the left upper lid, a dilated and fixed left pupil, with only lateral and rotary motion of this eyeball remaining, and evident paralysis of the left third nerve. The eyeball was tender to pressure. Examination of the fundus showed evidence of an old hemorrhage. There was no elevation of the left disk; vitreous opacity concealed the right disk. The right eye appeared exophthalmic, due largely to overelevation of this lid in attempts to see with the left eye. Spinal puncture released clear fluid under 18 mm. of pressure. The patient obtained some relief from spinal drainage, but continued to vomit and complain of frequent sharp throbbing pains over the left eye and attacks of chilliness or faintness during which the left arm felt numb. Neurologic examination showed hypesthesia, a sluggish abdominal reflex and a questionably positive Babinski sign and ankle clonus on the right side.

Diagnosis.—Cerebral aneurysm involving the left posterior communicating branch of the circle of Willis.

Operation.—Ligation of the left internal carotid artery was performed on April 26. There was prompt relief from the severe attacks of throbbing pain over the left eye. The patient complained that night of a fainting sensation in the region of the heart. On the following morning, slight elevation of the left lid was possible, and the signs of paralysis of the left third nerve gradually disap-

peared. She returned to light housework, with no recurrence of symptoms during the following eleven months.

SUMMARY

Cerebral aneurysm should be suspected in cases of spontaneous subarachnoid hemorrhage, particularly in persons under 40 years of age.

Anatomic peculiarities of the cerebral arteries and infectious emboli are significant in the etiology of cerebral aneurysm. Arteriosclerosis and syphilis rarely are the cause.

Focal neurologic signs frequently indicate the location of the aneurysm after rupture, rarely before, unilateral oculomotor palsy being the commonest localizing sign in this series.

Ligation of the internal carotid artery on the side of the lesion is indicated if recurrent hemorrhage occurs.

Cortical herniation into arachnoid granulations is of common occurrence in conditions with intracranial pressure. An unusual case of thrombosis of the motor cortex from herniation into large pacchionian bodies is reported.

NARCOLEPSY *

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Narcolepsy was first described in 1877 by Westphal and named in 1880 by Gélineau, who regarded it as a "new species of neurosis." It is a condition characterized by "the occurrence of attacks of irresistible sleep without apparent cause, and curious attacks on emotion, in which the muscles relax suddenly, so that the victim sinks to the ground, fully conscious but unable to move." This is the description of the condition as used in 1926 by Adie, who reported six personal cases and fifteen typical cases found in the literature, together with a review of the literature up to that date. In 1928, Wilson made a similar exhaustive study of the literature and reported five personal cases.

Most of the reported cases are to be found in the German literature, with only an occasional case in the English journals. That the condition is becoming more frequent is attested by the fact that Wilson was

* Submitted for publication, Aug. 6, 1930.

* From the Rees-Stealy Clinic.

* Thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Medicine.

on a constant look-out for a true case for twenty years before one appeared, and then he saw four cases within a few months. In 1925, Spiller had a similar experience, saying that "a few years ago it was exceptional for a neurologist with a large experience to see a case of typical narcolepsy. I have been impressed with the fact that at least three cases have come under my observation within a year." This study adds further evidence of an increased frequency of the condition. This may be partially explained by the fact that better histories are probably being taken and more careful observations being made, many of the previous cases being diagnosed as "profound somnolence," "hysteria" or "neurosis."

The term "narcolepsy," as suggested by Gélineau, has been much abused and used indiscriminately to include all variations and degrees of somnolence. This led to much confusion on the subject until Loewenfeld, in 1902, called attention to a double symptomatology in narcolepsy, viz., the combination of short intervals of apparently normal sleep with a peculiar transient condition of physical weakness and loss of power under the influence of emotion—the so-called cataplexy attacks. Adie and more recent authors have supported this view, and have succeeded in raising the term narcolepsy out of the chaos of somnolence to a definite clinical entity. Wilson, however, preferred to speak of the whole group as "the narcolepsies," narcolepsy being merely a symptom of a condition the origin of which is still unknown. This is in accord with Hughlings Jackson's views on epilepsy, and seems to be the more rational way of looking at the subject.

In this paper I shall present a report of forty-five cases of narcolepsy found in the records of the Mayo Clinic during the years 1919 to 1928, inclusive. Some of these are personal cases, but the great majority of them are taken from the files of the clinic. Letters of inquiry were sent to all patients who had not been at the clinic during the preceding two years to ascertain whether there had been any change in the character of the spells, or whether any form of treatment had been of benefit in relieving the distressing symptoms. All of the patients were submitted to the following examinations: general physical, neurologic, fundoscopic, Wassermann test, blood count and urinalysis. In addition, other tests were made as indicated in each individual case. For the sake of brevity, only positive observations are given, and if no mention is made of any of the studies enumerated, they are to be regarded as negative.

IDIOPATHIC NARCOLEPSY

CASE 1.—A civil engineer, aged 45, single, registered at the clinic on Jan. 28, 1919. At irregular intervals for eighteen years he had had transitory attacks, similar to "nightmare," in which he became drowsy and unable to move. He felt

that he was temporarily tied down, but if he was sitting he always tried to wriggle his way down in the chair in an effort to move. He never lost consciousness or control of his sphincters during these spells, which lasted from five to fifteen minutes, and which occurred at intervals of from one to six weeks. Occasionally, he had diplopia following an attack and felt weak in the knees. He was also troubled with sleepy spells in which he went to sleep while eating or while walking home from the office. Sleep was light and a slight jar easily awakened him. General examination showed some pyorrhea, but otherwise gave normal results.

This was the earliest case of somnolence approaching narcolepsy that I was able to find in the files of the clinic. The diagnosis made at the time of examination was "hysteria, on the order of narcolepsy," but in the light of present-day evidence, it seems to have been a true narcolepsy.

Case 2.—A man, aged 22, a student, single, registered at the clinic on July 17, 1920. Four and one-half years previously he had begun to go to sleep while studying, especially after the noon meal. He went to sleep while at the movies and once while driving a car, when he narrowly avoided an accident. While walking he often became drowsy and frequently had to sit down for a few minutes' nap. These attacks were always preceded by from five to ten minutes of lethargy, and continued for from five to thirty minutes. The sleep was always refreshing, and sometimes he dreamed during the attacks. Working outdoors and getting plenty of rest tended to lessen the number of attacks. When excited, his vision was often blurred, and frequently at an exciting part in a movie he would shake and tremble all over. If he pinched himself he could regain his control. General and neurologic examinations gave essentially negative results.

Case 3.—A man, aged 27, a clerk, single, registered at the clinic on Oct. 30, 1922. About two and one-half years before, while in the army, he had noticed that he was having difficulty in keeping awake while on duty, especially in the middle and latter parts of the day. One year before, he began to find himself going to sleep at the office, at the theater and even at dinner.

About one year before going to the clinic, he noticed that while crossing a small bridge which was infested with water-moccasins he would have to stop when he spotted one of these little reptiles, and that he would lose control of himself, shudder and be unable to move for a moment. At about the same time, when playing bridge he noticed that he would be all right while losing, but that if he were winning he would "go all to pieces," feel weak and be powerless to write the score or shuffle the cards. He then began having spells during which he would be momentarily unable to do things he wished to do; e. g., when he met people unexpectedly, he would be unable to move or speak for five or ten seconds. Two months before examination at the clinic, he began having spells which were evident to others, and in which he would suddenly lose control of himself; his head would fall forward onto the chest and he would mumble a few inarticulate words, but he was always fully conscious and aware of his surroundings. His mother always thought that he was asleep or even unconscious during these attacks, but the patient said not. The attacks occurred almost daily, lasted about fifteen or twenty seconds, cleared up spontaneously and left no residual effects. General and neurologic examinations gave negative results.

A letter received in March, 1929, nine years after the onset of symptoms, stated that there had been no change in the patient's condition.

CASE 4.—A housewife, aged 30, came to the clinic on Nov. 21, 1924. About fifteen years previously, while at school, she noticed that she would go to sleep while at lessons. This tendency had gradually progressed until she would go to sleep anywhere and under all sorts of circumstances, e. g., while sewing or playing bridge, while at a show or a football game, or while driving a car. The attacks, which occurred three or four times daily and lasted about fifteen or twenty minutes, were sometimes preceded by a transitory aura in which things seemed to "slip far away from her."

She also noticed that in emotional states she would suddenly feel weak, as though she were going to fall if she did not catch hold of something. She had never fallen, however, as she could partially control this feeling by a conscious effort of the will, but she was totally unable to control the sleepy spells.

Running parallel with the development of the diurnal drowsiness, she had had marked nocturnal restlessness. She kicked, tossed and thrashed about the bed, talking and laughing to such an extent that other people objected to sleeping in the same room with her. Her dreams usually fell into one of the following categories: (1) fear, in which she became terror-stricken about fire or the possibility of her child dying; (2) problems concerning her work, or some experiment which would never end; (3) mirth, when she laughed until she awakened herself, but could rarely recall at what she had been laughing.

Physical examination showed an absent tendo achillis reflex, resulting from a former sciatica, and a weakly positive Wassermann reaction, but otherwise gave negative results. She had had influenza in 1918, after the symptoms started.

CASE 5.—A man, aged 51, a Greek, who registered at the clinic on Dec. 16, 1924, had come to America twenty-five years before and worked as a cook. He had been married seven years later. He was successful in business, and up to 1917 had saved $14,000, which he had invested in German marks and lost; then, as he was unable to support his wife and family, the family returned to Greece. About seven years before coming to the clinic, he began to have sudden irresistible attacks of sleep. He would go to sleep while at work cooking, while eating or while riding in a street car. In fact, he would go to sleep at any time and in any place, and he had not been able to hold a steady job since that time. He slept for from five to fifteen minutes and awakened feeling refreshed.

Since the onset of the attacks of sleep he had noticed another peculiarity. While at work a waiter might order "ham and eggs" and he would get them all ready but forget to light the gas under them, or he might fry "hot cakes" instead, or when washing dishes he might dry a pan and hand it to the left as though there were someone there to receive it; while playing cards he might suddenly pick up the cards from the table and hand them to his friend; when his attention was called to his error he would immediately realize that he had made a mistake and would say, "Oh! excuse me," but he would have no real comprehension of what he had done. Even during conversation he might make an entirely irrelevant remark which would embarrass him considerably, but he was always able to remember the trend of his conversation and to resume it. Occasionally, while sitting quietly he would drop off to sleep and suddenly wake up realizing that he had made some movement with his hands; e. g., while sitting in the hall with his head resting on his hands, he dozed off to sleep and awakened suddenly to find that he was in the act of crossing himself. These attacks might occur as often as four or five times a day, or he might go through a whole day without one. The attacks lasted from one to five minutes, and he felt perfectly normal afterward.

For the past twenty years, nocturnal sleep had been restless and he awakened frequently. This condition had been more pronounced in the past few years, and

he admitted worrying about his financial losses and these peculiar spells. He always had more spells after a sleepless night, but remaining in bed for from twelve to fifteen hours would not prevent the attacks on the following day. He said that he had a "split sleep," by which he meant that part of his brain slept while part of it kept active; he had often been awakened with some thought on his mind, but on awakening fully and dismissing the thought he could go back to sleep immediately. Occasionally, he awakened at night with the feeling that there was someone in the room, and he had even arisen and looked about to reassure himself; sometimes he would feel certain that someone was stealing his blankets, but would be absolutely powerless to make the slightest move to prevent it or to help himself. Sometimes on awakening he would be totally unable to get up or even to move a muscle for a few seconds.

Apart from showing him to be 50 pounds (22.7 Kg.) overweight, examination gave entirely negative results.

Case 6.—A man, aged 21, a clerk, registered at the clinic on Feb. 12, 1925. In 1919 he had begun to notice that he would feel faint whenever he was in a warm room but that he would feel all right as soon as he went outside. The following year (1920) he noted a tendency to go to sleep while at school, and this increased in both frequency and severity until he was forced to give up school. He would go to sleep while at his meals, at a movie or in church. He then went to work in his father's grocery store, but would go to sleep while waiting on a customer. His father sent him outside one winter day to do some work and found him a few minutes later sound asleep, with the thermometer 20 degrees below zero. He would go to sleep while walking along the sidewalk, continuing to walk while asleep until he would bump into something or step off the walk. He often slept for from one to two hours if not awakened, and the amount of sleep obtained at night would make no difference in the number of attacks the following day.

Whenever he became excited or attempted to run after anyone, he would suddenly become weak in the knees and fall down; he was perfectly conscious, however, and able to get up again at once. While passing things at the table his arm would frequently relax suddenly and he would drop whatever he was holding. If he tried to fight an attack of sleep, his whole body would become shaky and tremble, his knees would become weak and he would fall. Nocturnal sleep was restless, and he turned and awakened frequently.

General physical examination gave negative results, and there had been no recent change in weight. The basal metabolic rate was plus 13. No history of an attack of influenza was obtained. From a letter received in March, 1929, it was evident that there had been no change in the patient's condition.

Case 7.—A man, aged 26, a clerk, who came to the clinic on Sept. 21, 1925, for the past fifteen years had had diurnal drowsiness, coming on especially after meals. This had progressed until he would go to sleep suddenly regardless of what he was doing, for example, while talking, walking or working. The sleep would last only a few seconds, and he would awaken refreshed. If he fell asleep while talking he would at times continue to talk, but incoherently and disconnectedly, or if he fell asleep while writing he would continue to write, but the marks would be meaningless.

He also had attacks of sudden muscular weakness when emotional. Sometimes when walking he would feel for a moment as though he could not take another step. While hunting, his arms would suddenly become weak and fall limply to his side as he raised the gun to shoot. He often felt that this weakness might become generalized, causing him to fall, but this had never happened.

Ever since the onset of the symptoms he had been troubled with nocturnal restlessness and insomnia. He had had "terrific nightmares" in which he would twist and toss about in bed, talking and crying aloud, and making such a disturbance that no one would sleep with him. He always felt more alert at night than in the daytime, and as a consequence did not go to bed until about 3 or 4 a. m.; he then slept soundly for about two hours, awakened and was ready to get up. After breakfast, however, the diurnal somnolence began.

Physical examination gave negative results. There had been no change in weight. The basal metabolic rate was plus 2. Although the symptoms began in 1910, the condition had been much worse since an attack of influenza in 1918. There was no evidence of a parkinsonian syndrome.

CASE 8.—A man, aged 29, an expert mechanic, single, who registered on Nov. 4, 1926, since the age of 15 had been troubled with attacks of an irresistible desire to go to sleep. These attacks came on at any time of the day and under all sorts of circumstances, but were more frequent when he was doing uninteresting and monotonous forms of work. He noticed this especially when compelled to watch the road before him continuously while driving a car; he narrowly escaped accidents on several occasions and had often stopped the car at the side of the road to take a few minutes' nap. He had gone to sleep standing up. He could not voluntarily ward off an attack, and a good night's rest would not lessen the frequency, but excitement and hard physical labor would help to overcome them. The attacks lasted for five or ten minutes, during which he slept soundly and sometimes dreamed.

At times, when angry or when making a witty remark he would feel a sudden and momentary "give" in the knees, or if he happened to be sitting, he would notice a similar phenomenon in the dorsal spine.

Both physical and neurologic examinations gave negative results. There had been no change in weight. The basal metabolic rate was minus 7. There was a history of influenza in 1918. A letter received in March, 1929, stated that there had been no change in the patient's condition after seventeen years' duration of the symptoms.

CASE 9.—A housewife, aged 28, registered at the clinic on Feb. 22, 1927. During the summer, 1921, while riding on a train, she had felt unusually drowsy and sleepy, but had thought nothing of it. During the winter, 1921-1922, sleepiness became much worse, and she experienced difficulty in keeping awake while teaching or doing things in which she was intensely interested. She was much concerned over the lethargic condition; she could not take an active part in social activities as she had formerly done, because she would drop off to sleep at inopportune moments in spite of all her efforts to keep awake; she had missed the context of a moving picture; she had been embarrassed at bridge, and on several occasions she had almost lost control of her car. Sleepiness was always worse after a meal or when she was tired and relaxed, and she would sleep as long as half an hour at a time.

Since marriage, in 1923, she had noted spells of weakness whenever some little occurrence affected her emotionally, such as meeting a friend unexpectedly; excitement or laughter would also bring them on. During these spells she was perfectly conscious, but felt helpless; the knees became weak, the eyelids drooped; the facial muscles relaxed, and she would be unable to utter a sound or even move the eyes. Vision became blurred, and the hands and feet seemed to go to sleep. She was accused by friends of taking "dope" and "too many cocktails." Nocturnal rest was disturbed by continual dreaming.

She had had influenza in 1918. Physical examination, including a fundoscopic examination, examination of the visual fields and roentgen examination of the head, gave negative results, as did also a neurologic examination. A gain of 24 pounds (10.9 Kg.) had been noted in the previous twenty-one months.

Case 10.—A man, aged 27, a cleaner and tailor, married, who registered on March 8, 1927, had had influenza in 1918, and was well following this until one day in 1921, when, while on duty in the navy, he suddenly became dizzy and fell over unconscious. Following this attack, which lasted about twenty-four hours, he became very nervous, dissatisfied, irritable and discontented; at this time he was told that his trouble was a result of the influenza. In 1921, also, he began to be troubled with spells of sleepiness and trembling. He was drowsy most of the time and would sleep as many as twelve hours a day. He would go to sleep soon after getting up, or while driving a car, and if he attempted to fight a sleepy spell he would usually have an attack of weakness or trembling.

In these attacks he would shake and tremble all over and would sink to the floor unless able to catch himself; although perfectly conscious he would be unable to say a word. The attacks would occur as often as thirty times a day and were transitory. They could be initiated at times by any form of excitement or by merely shaking hands; laughing always produced them.

General physical examination gave essentially negative results. The basal metabolic rate was plus 4. Apart from the attacks there was no evidence of a postencephalitic condition. A letter in March, 1929, stated that the spells of weakness were increasing in frequency and that the patient was unable to work.

Case 11.—A salesman, aged 45, married, who registered on May 17, 1927, for the previous three years had noticed an increasing desire to sleep. This had become worse in the past few months, and he would go to sleep whenever he sat down or when things were not particularly interesting. He had given up driving a car because of the frequent drowsiness. A few minutes' sleep was sufficient to refresh him. About the same time that the drowsiness started, he began to experience attacks of weakness. The first one occurred while he was dancing with his daughter; he became amused at some innocent remark and suddenly felt weak; his knees gave way, and he slowly settled to the floor, fully conscious but totally unable to help himself. Similar attacks of weakness had recurred frequently and were initiated by mirth or anger. The attacks were of a few seconds' duration and were usually associated with shaking and trembling of the extremities. The patient had an attack while in the clinic; he suddenly seemed agitated, the head slumped forward on the chest, the eyes closed, and he was unable to speak; he said afterward, however, that he was perfectly conscious of what was going on. He stated that he dreamed considerably at night and that these dreams were usually in the nature of a struggle or a fight with someone.

General physical examination gave negative results throughout. There had been no gain in weight. He had had influenza in 1918, and had had occasional headaches all his life.

Case 12.—A housekeeper, aged 43, single, who registered at the clinic on Oct. 14, 1927, for the past six years had been having periods of extreme lethargy which came on whenever she was inactive or whenever she fixed her gaze for too long a time on any one object. The attacks often occurred when she was in a crowd, when she was at a party or at church, or during conversation, and on some occasions she had been forced to lie down to go to sleep. She could fight an attack occasionally, but not often, and then only with great mental effort. After sleeping for from two to five minutes she awakened feeling refreshed, but the attacks

would recur as frequently as ten times a day. For the past one and one-half years she had noticed that she felt weak in the knees at times, especially whenever she tried to turn suddenly or started to run or walk.

The patient was obese, weighing 267 pounds (121.1 Kg.) and was 5 feet, 4 inches (162.6 cm.) in height. She had had influenza in 1922, one year after the trouble started. She also had had an operation for cholecystitis with cholelithiasis. Examination of the central nervous system and roentgen examination of the head gave negative results. A letter in March, 1929, stated that she had been benefited by taking thyroid tablets.

Case 13.—A garage worker, aged 19, single, registered on July 28, 1927. After returning to school, four years previously, the patient began to have brief periods of falling asleep for a few seconds in spite of the utmost concentration to keep awake. The attacks came on as often as six times daily, and their duration was from a few seconds to a minute. They occurred in the morning before breakfast in spite of eight or ten hours of sleep the previous night, or at dinner and while driving a car. It was because of a narrow escape from an automobile accident caused by going to sleep at the wheel that he came to the clinic. The attacks were more frequent during the first year than they had been since. He had also noticed momentary weakness of both upper extremities when laughing or when excited; for instance, if he saw a trout jump out of the water when he was fishing, he would get excited and experience a momentary weakness of the arms and hands, and to a lesser degree of the legs; on one such occasion the fishing pole slipped out of his hands and was lost.

Examination gave negative results throughout. There had been no gain in weight. He had had influenza in 1921. A letter in March, 1929, stated that there had been no improvement in the symptoms.

Case 14.—A salesman, aged 25, single, registered on Sept. 7, 1927. During the spring of 1927 he began to feel sleepy and would frequently go to sleep for a few seconds or a minute while reading a book. This tendency gradually increased until he began to go to sleep while at the wheel of a car. On several occasions he awoke to find himself on the wrong side of the road; once he was about to run into a railway train, and once he found himself in the ditch. Although he fully realized his danger he was unable to prevent himself from going to sleep, and finally, six weeks previous to the examination, he was forced to give up his position and driving a car. For the past three months, whenever he laughed or became angry the knees would suddenly give way under him for a second, but he had never fallen to the ground as he could always catch himself in time.

The patient had had a penile lesion six months before the present condition started, although he had never had a positive Wassermann reaction or any secondary manifestations of syphilis. He had never had influenza, and physical examination gave negative results throughout.

Case 15.—A farmer, aged 22, single, who registered on July 5, 1927, about two and one-half years before had noticed that he would go to sleep while reading; he attributed this to an insufficient amount of sleep at night, so for one week he averaged nine hours' sleep per night, with no improvement in the tendency to sleep during the day. A year and a half later, he began to go to sleep while at work, on a wagon, milking, or even while driving a car. There was a moderate degree of improvement for three months during the winter, but in the spring the symptoms returned. At the time of examination he would go to sleep as often as twelve times a day, sleeping for from one to fifteen minutes, and after waking would feel bright and refreshed. During these sleeping spells he often continued to perform

automatic movements; e. g., if he were walking behind the harrows or a plough, he would continue to walk and would awaken only when the horses stopped at the end of the field; he believed that he had walked 50 or 60 yards under such conditions. The only prodromal symptom he had was a feeling of heaviness over the eyes. By walking or smoking vigorously he could sometimes, but not always, abort an attack.

On two or three occasions during the spring, he became weak in the knees while watching the antics of a pet dog; these attacks were never severe enough to cause him to restrain his mirth.

The only positive observations at a physical examination were a slight horizontal and vertical nystagmus, and two infected teeth. There had been no gain in weight. Roentgen examination of the head and sella turcica gave negative results. He had had influenza in 1921. A letter received in March, 1929, stated that there had been no change in the condition.

Case 16.—A farmer, aged 21, single, who registered on May 31, 1927, during the summer, 1925, had noticed that there were times when he could not keep awake. The first attack came on while he was on his hands and knees weeding the garden, when he suddenly dropped off to sleep, his muscles relaxed and he fell forward, the sudden fall awakening him. At first these attacks occurred once or twice a week, each one lasting from one to five minutes. In the fall, 1925, he went to a university, but would go to sleep from four to six times during a lecture hour and whenever he sat down to study; he finally had to resort to studying while walking around the campus. In March, 1926, he fell asleep while driving and drove the car off the road, striking a telephone pole. The drowsiness was more intense from 11 a. m. to 2 p. m., and at 4 p. m.; five minutes' sleep was sufficient to refresh him. Going to bed early had no effect on the number of attacks on the following day.

Since the condition had started, the patient had noted a momentary relaxation of muscles whenever he became emotional; if he laughed or became angry he felt weak all over. If he expected this muscular weakness he could sometimes ward it off. He was practically incapacitated, and could not hold a job satisfactorily. Sleep both during the day and at night was restless and disturbed by dreams.

Examination showed a decrease in the deep tendon reflexes, but otherwise gave negative results. There had been no gain in weight, and there was no history of influenza. The patient's mother had suffered from two nervous breakdowns, and a maternal aunt was a patient in a state hospital.

Case 17.—An insurance salesman, aged 46, married, who registered on Dec. 21, 1927, in 1917 had first noticed attacks of drowsiness and a tendency to go to sleep whenever he was in an environment of monotony. While sitting quietly in a chair, riding a horse or driving a car, he would drop off to sleep in spite of all efforts to keep awake. Sometimes while driving he would have to stop, get out and walk around in an effort to postpone the sleep temporarily, but it always overpowered him sooner or later. From fifteen to thirty minutes of sleep was sufficient to refresh him.

In 1923, he began to have another kind of attack. Whenever he reached the climax of a funny story that he was telling, or was just about to sell someone life insurance, or at an exciting moment during a card game, his whole body would suddenly become weak and limp. During an attack, if standing, he maintained the erect position with difficulty; the eyes closed and he was unable to speak although he understood everything that was said. These attacks finally became so severe that he would fall down, totally flaccid and helpless, but per-

fectly conscious. All emotions did not cause this weakness, as was evidenced by the fact that only a year before in a fit of anger he cursed a man for over an hour without being affected; also he could laugh when someone else told a funny story, without any effect.

The patient had an attack during the examination. He asked permission to smoke, saying that smoking sometimes helped him. The examiner offered him a cigaret which he took; he reached for a match but immediately dropped it; the cigaret fell from his fingers to the floor; he slumped over in the chair with the eyes closed and mumbled some inarticulate words. During this attack the pupils were small, but equal, the lower jaw trembled and the chin and lower facial muscles twitched. All deep tendon reflexes were markedly diminished or absent, and the muscles were soft, flabby and toneless. Unfortunately, the Babinski test was not made. Within ten minutes he had regained composure, and the reflexes had returned to normal. He said that he was fully conscious all the time and that it was not the generosity of the gift of a cigaret but rather the attempt to express his gratitude that precipitated the attack.

For the past twenty years, the patient had been a restless sleeper, tossing and twisting about in bed, talking, cursing and crying aloud so that neighbors could hear him. In 1924, the dreams became "terrible and horrible," and he dreamed that he was being chased by snakes, monsters and all sorts of wild animals. They were all terrifying experiences, and would often awaken him by their horrible and gruesome nature; this emotion of fear would persist into the waking state and precipitate a spell of weakness.

Examination gave negative results throughout, except for the attack during the examination. The patient was 6 feet, 2 inches (188 cm.) tall, and weighed 240 pounds (108.8 Kg.). There was no history of influenza.

CASE 18.—An oil-well employee, aged 29, married, registered on Jan. 13, 1928. Three years previously he had noticed the gradual onset of drowsiness in which he would go to sleep at any time, especially whenever he sat down in the house, in a street-car or at a moving picture. He had had three automobile accidents due to going to sleep while driving. He had also gone to sleep while standing up, and had fallen asleep in the waiting room of the clinic. The attacks occurred daily, and he usually slept from fifteen to thirty minutes, awakening spontaneously and feeling perfectly refreshed.

One year after the onset of the sleeping attacks he began to have a peculiar, sudden, weak, quivering feeling about the face and especially around the mouth whenever he became amused or angry. During the past year this weakness on emotion had spread to involve the whole trunk, and he had fallen to the ground on five or six occasions.

For the past eighteen months he had had another kind of attack during the day in which he became dyspneic, a glimmer appeared before the eyes, the body became limp and weak, and he would fall if he could not lean against some solid object. He was dimly conscious of what was going on, but was powerless to speak or move. He usually had a fine tremor of the hands and arms, associated with slight convulsive movements of the arms and face, and less frequently of the legs. These attacks occurred almost daily and seemed to be more prone to come on while he was sitting or lying down. They lasted for from a few seconds to a minute and sometimes could be inhibited by a long deep inspiration. He never had any loss of sphincter control, and had never bitten the tongue or injured himself during the attacks. Nocturnal sleep was disturbed by nightmares, and he often had the feeling that someone was trying to crawl in at the window; during

this his whole body trembled and shook and he felt powerless to move or to speak. A light touch from his wife would be sufficient to stimulate him and to bring him out of such an attack.

Both physical and neurologic examinations gave negative results. There had been no increase in weight. He had had influenza in 1918. The patient had a philosophical way of looking at the trouble, and did not let it interfere with his enjoyment of life.

Case 19.—A man, a ticket clerk, aged 26, married, who registered on June 7, 1928, one year previously had noticed that his knees would become weak and tend to give way whenever he was telling a funny story or laughing. At first this occurred about once a week and was not severe enough to cause him to fall. Two months before examination, while talking and laughing with his wife and some friends on his return from work, the legs suddenly became weak and he fell to the floor, fully conscious but unable to move or speak for thirty seconds; he then got up without help and felt as usual. The next day he had three similar attacks and went to a hospital for three weeks. During this time he improved, but after returning home the spells of weakness continued, until at the time of examination they occurred several times daily.

During the six months previous to examination, he had noticed an increased drowsiness and tendency to go to sleep, so that he found himself going to sleep while at work; he had also driven a car into a ditch on two or three occasions during sleeping spells.

The previous history was without significance, and examination gave entirely negative results.

Case 20.—A manufacturer, aged 31, married, registered on Feb. 10, 1928. Two years before, he began to notice that he would drop asleep while at moving pictures, directly after meals or whenever he attempted to read. This tendency gradually became more pronounced, and during the past year he had fallen asleep while at the office, while standing up or while driving a car. His father had noticed him asleep at his desk with a pen poised in his hand preparatory to writing. He slept for from five to fifteen minutes and awakened spontaneously.

About once or twice a week during the past six months, when laughing he had suddenly felt weak in the knees as though someone had struck him a blow in the popliteal space. This was accompanied by a "gone" and sinking feeling, but was of momentary duration and he had never fallen.

The patient came from a normal, healthy family; both parents, three brothers and two sisters were alive and well. He was well established in business and was successful. He had attended a university and would have graduated had he not entered the army in 1917. His habits were regular and steady, with moderate sexual promiscuity during and after his attendance at college. Three years previously, after a brief acquaintance of three months, he married the only daughter of a domineering mother who had failed to instruct her in sexual matters. The daughter was frigid and would not allow herself the normal feelings of sexual intercourse; she became pregnant two months after marriage. This so angered the mother that she insisted, against the husband's wishes, on taking the daughter home, where an abortion was performed. The wife then returned to her husband, but would not allow any sexual activity for the next two years, still cherishing a profound mother-love complex and spending most of her time at the mother's home. The mother-in-law hated and despised not only the patient, but all his family as well, and never failed to make a caustic remark whenever the opportunity presented itself. Under such circumstances the patient's trouble began.

Examination revealed a well developed man, 6 feet (182.9 cm.) tall and weighing 206 pounds (93.4 Kg.); he had gained weight during the past two years. Aside from this, the examination gave essentially negative results.

CASE 21.—A rural mail carrier, who registered on Nov. 12, 1928, had been well until five years before, when he noticed that he would be unduly sleepy whenever he was in a warm room, especially in the early morning, about noon and immediately after a meal. He usually went to work about 3 a. m., but by 4 a. m. would be so sleepy that he would have to sleep for a few moments; after this he would feel perfectly refreshed until noon. At times an intense drowsiness would occur while he was eating or driving a car, and he would go to sleep unless he could immediately start some vigorous exercise or wash his face in cold water. He was not always able to resist the attacks, however, and under such conditions he had gone to sleep while driving his car.

Shortly after the attacks of drowsiness started, he began to have spells of weakness. The first attack occurred while he was working, and consisted in a gradually generalized muscular weakness, in which the muscles relaxed and he fell to the ground but was fully conscious. In a minute, strength had returned and he was able to get up, feeling all right. These attacks occurred as often as once or twice an hour, and were gradually becoming more frequent. The unexpected meeting with a friend and the sight of a vicious dog or of a snake would always bring on an attack of weakness. During the attack he usually lost control of the tongue, the eyelids drooped and the lips quivered. Sometimes an attack could be aborted by increasing the tension of the muscles of the legs or the tongue, but this took a great deal of mental energy and was very difficult. Some of the attacks in which he collapsed to the floor were followed immediately by, or passed into, a sleeping attack.

The patient was 5 feet, 8 inches (172.7 cm.) tall and weighed 164 pounds (74.4 Kg.), which represented a gain of 20 pounds (9 Kg.) since the onset of the trouble. Examination gave essentially negative results in other respects. He had had influenza during the epidemic of 1918.

CASE 22.—An insurance agent, aged 32, married, who registered on June 11, 1928, had begun to notice sudden attacks of sleepiness while in the army in 1918. He would feel tired and drowsy for about five minutes and then go soundly to sleep for from one-half to one and one-half hours. At first these attacks occurred only four or five times a year, but in the four years previous to examination they had occurred as frequently as four or five times a day and under all circumstances. He had had four automobile accidents caused by going to sleep while driving.

About the same time he began to have attacks of momentary weakness, during which he would have to catch hold of something to prevent falling. He had fallen to the ground many times, not losing consciousness, but was able to get up again without delay. While at the table his arm often became weak and he would drop whatever he was holding. During these attacks he sat in a slumped posture, with the eyes closed and the head nodding on the chest, and was unable to answer questions or respond in any way, although he said that he knew and heard everything that went on. He had an attack which lasted for fifteen minutes during the examination. These spells of weakness came on whenever he laughed or was amused or excited, and he also felt that laughing and excitement tended to induce a sleeping attack.

For the past five years, nocturnal sleep had been disturbed by unpleasant dreams and nightmares, during which he often carried on a conversation with an

imaginary person; he was perfectly convinced in his own mind, however, that he was having a nightmare. In the dreams he often saw cats, rats and snakes in bed with him. Occasionally he dreamed during a sleeping spell in the daytime.

Physical examination revealed a well developed man, 5 feet, 9 inches (175.3 cm.) tall and weighing 156 pounds (78.8 Kg.), about 10 pounds (4.5 Kg.) below his average weight. The basal metabolic rate was minus 14 and minus 17 on two occasions, although the patient did not present a clinical picture of hypothyroidism. Examination of the eyes and roentgen examination of the head gave negative results. For the past ten years he had had frequent frontal headaches, but he did not think that they were in any way related to the other attacks. Previous to this, his health had always been good and he had never had any acute condition simulating influenza.

CASE 23.—A man, aged 26, a school teacher, single, registered on Aug. 9, 1928. Five years before, in 1923, while still a student, he had felt listless and tired during the latter part of the year and had been unable to sleep well at night. Following this, while on a fishing trip he slept more than usual and suffered from general malaise with various indefinite pains and aches throughout the body; since he lived in a malaria-infested district, he empirically took quinine and went to bed for three days; he then felt better, got up and went downtown. As he walked along, however, his legs suddenly gave way temporarily and he had to catch himself to prevent falling. This occurred several times without any special emotional stimulus and was followed in about two weeks by sudden sleeping spells of from one to fifteen minutes' duration. During the summer, 1923, he would go to sleep whenever he was sitting around without anything in particular to do, and the spells of weakness became so much worse both in severity and in frequency that his parents would not allow him to go to town alone. By September, 1923, he had improved sufficiently to go back to school, but was unable to keep awake during class. In 1924, he began to teach school but was still troubled by frequent sleeping spells of momentary duration; while conducting a class he would frequently sleep for just a second or two and feel refreshed afterward. He had even gone to sleep as the clock started to strike and had awakened before it finished striking. When he timed himself by a watch he found that he usually slept for from fifteen to forty-five seconds. On several occasions he had driven a car into a ditch while sleeping.

The spells of weakness had been present throughout the trouble, and were at their maximum severity in 1923. They were initiated by laughing or anger or by meeting a friend unexpectedly, when he would find himself unable to shake hands, the arm having fallen limply beside the body. General muscular relaxation occurred for a period of from ten to fifteen seconds, although the head, shoulders and muscles of the arms were chiefly affected; the shoulders slumped forward and downward, the head fell forward on the chest, and the eyes closed. He had never been unconscious. On two or three occasions while in bed, just as he was about to go to sleep, he had suddenly become very weak and faint and thought that he was going to die. He was fully conscious, but totally unable to move a muscle; he could talk, however, and had called to his mother. As soon as she touched him his strength returned and he was himself again.

About four months previous to examination, nocturnal sleep became much disturbed by nightmares and dreams in which he was always fighting (and he never had had a fight in his life). He often saw many long-tailed devils with spears and was afraid that he was dying. At the time of the examination his greatest trouble was nocturnal insomnia, and it was for this that he had come to the clinic.

Aside from a gain of 32 pounds (14.5 Kg.) since the onset of the trouble, examination gave negative results. He had had influenza in 1918, but the present trouble started insidiously during 1923.

NARCOLEPSY AFTER PROBABLE EPIDEMIC ENCEPHALITIS

The protean nature of the symptoms of a postencephalitic condition are such that no one who has seen a large group of these cases would question the possibility of the occurrence of symptoms similar to or even identical with those of narcolepsy. That such patients do show both attacks of sleep and cataplectic attacks identical with those occurring in idiopathic narcolepsy is shown by the following cases, as well as by those reported by Symonds, Spiller and others.

CASE 24.—A farmer, aged 36, married, who registered on June 22, 1922, had been well until he had influenza in November, 1918, during which he had high fever and was delirious for one night. In June, 1919, he began to notice the onset of drowsiness, and by December this had progressed to the severity present at the time of examination. During the day he usually had three or four attacks, in which he became so sleepy and drowsy that he could not do otherwise than stop whatever he might be doing to lie down and sleep. He slept for from fifteen to thirty minutes, awakened spontaneously and felt refreshed. He had gone to sleep while riding on the binder or plough, while at church, while typewriting or while eating meals. When driving a car he might have to stop, get out and lie down to sleep for a while before he could continue on his way. He taught a class in school during the winter, and frequently, after giving the pupils a lesson, he would have to leave the classroom to sleep for a few minutes. By sleeping regularly at 9 a. m., noon and 7 p. m., he could get through the day successfully. Nocturnal rest was disturbed by unpleasant dreams of a wild and exciting nature, causing him to toss about in bed, call out and talk a great deal. This restlessness usually started about four hours after retiring and continued until morning.

In 1920, he began to have other attacks. When he attempted to throw anything, he was immediately seized with a sense of faintness and weakness which commenced in the legs and passed in a wave over the whole body. During this time he felt weak and shook and trembled all over; his mind became somewhat cloudy, and, although he was not unconscious, he had difficulty in speaking distinctly. These spells lasted for from thirty to sixty seconds, and at first occurred only when he attempted to throw something. Later they occurred whenever he became earnest in speaking or whenever he lost his temper, but never when resting quietly. They would often check him in some sudden emotion or action; e. g., once when he attempted to beat a horse, he was suddenly seized with a spell of weakness in which the head sagged forward, the muscles gave way, and he was forced to lean up against the horse for support; when the spell had passed off and his mind cleared, he was able to get up and beat the horse as he had originally planned.

The patient had an attack during the examination, while talking with some emphasis. Another attack was induced by attempting to throw a towel, while a third one failed because he said his mind was on it, maintaining that the production of the attack seemed to depend somewhat on his state of mind. He could, seemingly, fortify himself against an attack if he had sufficient warning.

Description of Attack.—During the attack, the whole body suddenly relaxed and wilted, the eyelids drooped conspicuously, the pupils became smaller, the face

flushed, the lips became cyanotic, and the whole face quivered with a fine rapid tremor. The hands fell limply to the sides and trembled in a manner similar to the trembling of the facial muscles. The whole expression and appearance was that of having been stunned by a blow on the head or of one half-intoxicated. While on his feet, the trunk drooped forward, the knees gave way, and, catching the couch with his hands, he stumbled onto it and sat down. He answered questions slowly, obeyed commands and seemed fully conscious, but he was unable to speak audibly or distinctly. Then the face paled, the ptosis disappeared, and he was himself again, the whole attack having lasted for from thirty to sixty seconds.

Examination revealed a slight left homonymous notch in the perimetric fields of vision, with normal fundi. The basal metabolic rate was normal, and roentgen examination of the head gave negative results. There had been a gain of 25 pounds (11.3 Kg.) since the onset of the condition. Neurologic examination gave negative results, and there was no other evidence of intracranial disease.

Case 25.—A man, aged 32, a miller, married, who registered as a patient at the clinic on Nov. 12, 1923, had been well until he had influenza in March, 1920, during which he had a temperature of 102 or 103 F. for a period of two weeks. In May, 1920, he began to have attacks of drowsiness, coming on at long intervals, and usually when he was sitting down. These spells gradually became more frequent until they occurred daily, coming on whenever he sat down for a few moments, whether at home, in church or at a show. They might come on while he was working, and he would have to stop to lie down and sleep for from fifteen to twenty minutes. He awakened spontaneously, felt refreshed and could go on with his work. At first he was able to ward off the attacks by walking, but later this became impossible.

Six months before going to the clinic, he noticed attacks of sudden loss of power in the arms and legs, with inability to speak, whenever he became excited or emotional. During these attacks he usually caught hold of something or leaned against the wall for support, fully conscious, but unable to talk or help himself. The attacks lasted about thirty seconds. A letter from this patient in March, 1929, stated that there had been a diminution in the frequency of the attacks of sleep and a complete cessation of the spells of weakness during the preceding six months.

Description of an Attack.—The patient was in the middle of the room walking on the tips of the toes when a sudden vacant stare appeared on his face, the eyes rolled up, and all the muscles seemed to relax and become weak, although he continued to walk until he reached the table, where he stood, supporting himself until the attack was over, unable to speak but conscious of all that was going on around him. The attack lasted about forty seconds. General examination gave negative results; there was no evidence of Parkinson's syndrome.

Case 26.—A farmer, aged 24, married, who registered as a patient at the clinic on Jan. 15, 1924, had been well until he had influenza in July, 1913, following which drowsiness became a marked symptom. He went to sleep while eating or while driving a car or a team of horses and would sleep for ten or fifteen minutes before he awakened spontaneously, feeling refreshed. For six months he had been more nervous and irritable than formerly and had peculiar "muscles jerkings," in which he suddenly lost power in certain groups of muscles. These attacks were brought on by emotion, excitement or physical exertion and might affect any part of the body; the head might be pulled back or the arms suddenly give way, causing him to drop whatever he might be carrying, or the legs might give way under him, allowing him to fall to the ground, conscious but helpless for a few seconds. Once,

while driving along the road accompanied by a brother, he saw something bright and shining in the road and got out to pick it up. When he saw it was a gold watch, his legs suddenly gave way and he fell in a heap in the middle of the road, totally helpless. The muscles twitched and jerked for a few seconds, then strength returned and he was able to get up before the brother could get to him to offer assistance. At no time had he ever been unconscious. Nocturnal sleep was restless, and he was easily awakened.

He had gained 10 pounds (4.5 Kg.) since the trouble began. He was given desiccated thyroid, which seemed to help him temporarily. A follow-up letter in March, 1929, indicated no change in the condition.

Case 27.—A woman, aged 24, a school teacher, single, who registered on Sept. 24, 1927, had been well until 1920, when she had an attack of influenza and was in bed for one week. In the early part of the present illness she had difficulty in getting to sleep at night, but toward the end she became unusually drowsy and somnolent, sleeping most of the time and going to sleep while engaged in conversation or while eating meals. This persisted until by the time of examination at the clinic she was falling asleep under all conditions, at meals, while at the moving pictures or while driving a car. Whenever she wished to go out she took two or three hours' sleep beforehand and in this way was able to keep awake for two or three hours at a time. The sleeping attacks occurred many times daily, and she felt drowsy and tired most of the time.

One and one-half years before going to the clinic, the patient began to notice sudden spells of weakness affecting the extremities whenever she laughed or experienced any undue emotional excitement. At first these consisted of a sudden, transitory weakness of the arms and legs, but later the weakness became more marked, causing her to drop whatever she was holding in her hand, or to fall to the ground if she was unable to catch hold of something for support.

General examination gave negative results, except for fibrous tonsils and one infected tooth. She was given sodium cacodylate, 1 grain (0.065 Gm.) intramuscularly, daily for ten days and advised to take as much rest as possible. A letter from the patient in December, 1928, reported temporary improvement in the spells of weakness, but otherwise no change.

Case 28.—A barber, aged 22, single, who registered on Jan. 17, 1928, in June, 1926, had fallen asleep while working and had had to be awakened by his employer. Ever since, he had been troubled with sleepy spells during the day, restlessness at night and spells of weakness under excitement. The sleepy spells became so troublesome that he was forced to change his occupation to that of mail carrier.

Excitement affected the patient peculiarly, and it was difficult for him to describe the sensations under such conditions. He thought that he acted "hysterical-like," because sometimes while passing things at the table, if he became embarrassed, his arm would shake and jerk slightly. On two or three occasions while laughing he had fallen to the floor on account of sudden loss of strength in the legs. Nocturnal sleep was always restless, and he rolled around the bed, dreamed a great deal and awakened frequently. He required between twelve and fourteen hours of sleep daily to keep going, and if deprived of nocturnal sleep the diurnal attacks were much more frequent.

The previous history was without significance, except for a severe attack of measles in 1918, during which he was very ill, with high fever and delirium for two days; he was in bed for about three weeks. There had been no increase in weight, but the patient was more irritable than formerly. He showed a dull,

sleepy expression and a lateral nystagmus. There was a background of vagrancy, restlessness and inadequacy, with a stepmother situation at home. A diagnosis of a probable mild encephalitic residue was made.

Comment.—Influenza, or more accurately epidemic encephalitis, has always been regarded as an etiologic factor in the production of any peculiar nervous condition that was not clearly understood. This has been especially true since the world-wide epidemic in 1918, but to assume that most of the cases of narcolepsy are sequelae to a previous encephalitis would be erroneous, as the following analysis shows.

Of the forty-two cases presented, there were seventeen in which there was no history of an acute illness which simulated either encephalitis or influenza in any way, and seven in which influenza occurred after the onset of narcoleptic symptoms; that is, in twenty-four of the forty-two cases influenza played no etiologic rôle. Cases 24 to 28, inclusive, may be postencephalitic, as they followed directly an acute infection which might be interpreted as encephalitis; the patients in the remaining twelve cases gave a history of having had influenza on an average of five years previous to the onset of symptoms.

Ziegler showed that 15.2 per cent of 752 patients with postencephalitic parkinsonian syndromes gave no history of encephalitis. If this percentage is applied to the 17 cases reported here in which there was no history of influenza, the number of probable postencephalitic cases would be increased only to 8.5 or only 20 per cent of the total number of cases. Therefore, it does not seem probable that influenza played as important an etiologic rôle as might have been supposed on first analysis.

NARCOLEPSY FOLLOWING ACUTE INFECTIONS OTHER THAN ENCEPHALITIS

Case 29.—A single woman, aged 29, registered as a patient at the clinic on Sept. 13, 1920. Ten or twelve years previously she had had a severe attack of pneumonia which lasted about six weeks, during a part of which she was unconscious and delirious. Following this illness she noticed an ever-increasing drowsiness and desire to sleep. She would go to sleep while at church, at lectures, at the theater or even at the table while eating. No matter how interested she might be she would go to sleep, and a good night's rest would not lessen the frequency of the attacks. She awakened frequently at night; sleep was restless, and at times she felt that there was someone in the room.

At irregular intervals, sometimes daily, sometimes less frequently, certain groups of muscles would suddenly relax and give way temporarily; e. g., the knees might give way and she would sink to the floor; the muscles of the neck might relax and the head nod forward, or the arms might suddenly become limp and flaccid, so that she would drop anything she might have in the hands. These attacks always accompanied a spell of anger or mirth, and might be repeated in quick succession. She never lost consciousness and usually recovered immediately.

General and neurologic examinations gave essentially negative results.

CASE 30.—A farmer, aged 18, who came to the clinic on Sept. 3, 1926, had always been well until May, 1925, when shortly after an attack of parotitis he began to notice spells in which his head felt warm and heavy, and he became so drowsy that he would have to go to sleep. After sleeping for ten or fifteen minutes, he would awaken spontaneously, feeling perfectly refreshed. At first these attacks came only once a day, but at the time of his visit to the clinic they were occurring three or four times daily. They occurred while he was eating, talking or driving a car, and he had driven an automobile into a ditch on two occasions. Sometimes, while doing manual labor, he could continue to work through the attack, which usually lasted about half an hour, although his movements would be more or less automatic as he would not know exactly what he was doing. He would have to sleep later on, however. If he laughed heartily his whole body would tend to give way under him, and he would fall to the floor or be compelled to sit down as soon as he felt the weakness coming on. Nocturnal sleep was restless, but nothing was said about dreams.

A general physical examination showed his weight to be 186 pounds (84.4 Kg.), a gain of 36 pounds (16.3 Kg.); his weight later increased to 200 pounds (90.7 Kg.). The systolic blood pressure was 110, and the diastolic, 72; the pulse rate was 72. He had had influenza in 1918.

CASE 31.—A school teacher, aged 22, single, who registered on June 13, 1923, began to experience sleepy spells about three weeks after an attack of scarlet fever, four years before. These would come on at any time, while she was teaching, eating or driving a car, and she was forced to give up driving for fear of accidents. She required a great deal of sleep at night, often sleeping from 7 p. m. to 7 a. m.; even then she would go to sleep again before breakfast. She would sleep for five or ten minutes and awaken refreshed. She could fight the impulse to sleep for a short time, but eventually it overpowered her in spite of anything she would do.

One year after the sleeping spells started, she began to have nervous spells during which she suddenly felt weak all over and trembled inwardly. These attacks were initiated by some emotional excitement, when she laughed, or at the critical moment in a moving picture, or when some one spoke to her suddenly while on the street, and especially when she made a quick movement to shake a youngster at school. The attacks were characterized by sudden, staccato, forward-jerking movements of the shoulders, trunk and elbows, associated with jerking and weakness of the knees, and were of such severity that she was sometimes forced to lie down on the floor until the attack passed off. During an attack the eyelids drooped, the jaw dropped open, and the head slumped forward on the chest, while the arms and hands hung limply beside the body. She was unable to speak or make a sound, but was fully conscious. The attacks usually lasted for from five to ten minutes, and she felt well afterward. Occasionally, the shaking of the shoulders looked like a laughing spell, and she had frequently passed it off as such. The patient was practically incapacitated on account of the trouble. She did not care to go out because of the embarrassment occasioned by going to sleep and by the development of a spell of weakness if she became excited or laughed. Sleep at night was restless and disturbed by many dreams.

With the exception of influenza in 1919, the patient had been well until the attack of scarlet fever, which was followed by desquamation three weeks previous to the onset of the present complaint. She had gained 15 pounds (6.8 Kg.) since the trouble began. Physical examination gave negative results.

NARCOLEPSY PROBABLY OF HYPOTHALAMIC ORIGIN

CASE 32.—A telephone operator, aged 35, single, registered at the clinic on Oct. 6, 1926. Three weeks before, when working as a switchboard operator, she was suddenly seized with a spell of weakness while laughing. During this attack she became very weak, trembled and shook, became pale and was unable to speak, but was conscious and knew everything that was going on about her. The attack lasted about a minute. Since then, similar spells had recurred as often as several times a day or as infrequently as once a week. Occasionally, she had had diplopia and vertigo with the attacks. The attacks were always brought on by emotion and excitement. She had always been drowsy and sleepy, and she fell asleep several times while waiting at the clinic; she was much more concerned about the spells of weakness after emotion, however.

The patient gave a history of having had influenza in 1918. In the family history it was revealed that her brothers and sisters were all large, heavy people. One brother, aged 21, weighed 240 pounds (108.8 Kg.); a brother, aged 38, weighed 180 pounds (81.6 Kg.); a sister, aged 31, weighed 157 pounds (72.1 Kg.); a sister, aged 33, weighed 200 pounds (90.7 Kg.). The mother weighed 182 pounds (82.6 Kg.) and the father, who developed diabetes, weighed 230 pounds (104.3 Kg.). Three years before, the patient had had an unfortunate love affair, with two induced abortions, to which she attributed her present trouble. She was of a very nervous and emotional type.

Physical examination showed her to be 6 feet (182.9 cm.) in height and markedly overweight, weighing 319 pounds (144.7 Kg.), 100 pounds (45.4 Kg.) of which she had gained in the past three years. The systolic blood pressure was 104 and the diastolic 76; the pulse rate was 92. The basal metabolic rate was minus 17. Roentgen examination of the head and a neurologic examination gave negative results.

The patient was given desiccated thyroid extract until the basal metabolic rate became plus 1, but with no improvement in the attacks. A letter in March, 1929, stated that she was still overweight and that she was unable to work on account of the sleeping spells which occurred two or three times daily. She had also continued to have the spells of weakness.

CASE 33.—A man, aged 27, married, who registered on Aug. 10, 1927, in June, 1924, had had severe "ptomaine poisoning," and shortly thereafter began to have sleepy spells. He was living in the south at that time and attributed it to the hot weather. He would sleep twenty hours a day if left alone. His wife would get him up and start him for the office, but after going two or three blocks he would often return home and go back to sleep; if he continued on to the office, he promptly went to sleep as soon as he arrived there. He would go to sleep while walking or driving a car, and would sleep until he awakened spontaneously or was awakened by driving into a ditch. About the same time that the sleeping spells started, he noticed that any emotion would cause him to become weak in the knees. If he became angry or laughed, or if he met a policeman or a strange dog on the street, the knees would suddenly give way and he would have to catch hold of something to prevent himself from falling.

The patient stated that his personality had changed since the onset of the trouble, and that he had become an "awful liar and coward," maintaining that he lied just for the fun of it, and that he was afraid of anything and everything. He became depressed about his condition and had contemplated suicide. Associated with this condition there had been increased appetite and thirst, with a rapid increase in weight of from 40 to 50 pounds (18.1 to 22.7 Kg.).

Examination showed the patient to be 5 feet, 9 inches (175.3 cm.) tall, and to weigh 202 pounds (91.6 Kg.). The systolic blood pressure was 118, and the diastolic, 86. The pulse rate was 68. The basal metabolic rate was minus 2. Urinalysis showed a specific gravity of 1.027 and was negative for sugar. Roentgenograms of the head and sella turcica were normal, and the fundi and fields were normal. There was a history of influenza in 1918.

The patient was given roentgen therapy to the pituitary region. A report received in October, 1928, stated that there had been some improvement, and that he was able to work half a day at a time, but that unless he had roentgen treatments every sixty or ninety days, he would get a "crazy" feeling in the head.

CASE 34.—A farmer, aged 34, married, who registered on Dec. 7, 1927, for the past four or five years had been troubled with headaches, sleepy spells, spells of weakness and general slowing up of all physical and mental activities. The sleepy spells came on at any time, while he was in the field working, while sitting quietly in the house or while eating meals. He usually slept for about five or ten minutes before waking spontaneously. If someone shouted at him, he would suddenly lose all strength in the extremities and fall to the ground, or if he attempted to run he would fall down unless he could catch hold of something for support. Sudden excitement or exertion would bring on a spell of weakness in which he would be powerless to move; vision would become blurred and speech would become thick and inarticulate.

Coincident with the symptoms recorded, he had had daily right, supra-orbital and temporoparietal, duli, aching headaches. During this time his weight increased from 175 to 247 pounds (79.4 to 112 Kg.), and he became mentally dull, inefficient and forgetful. Sexual power and libido were markedly decreased. There was no change in size of gloves, hat or shoes. He had had influenza in 1918, 1922 and 1925, and had been well until after the attack in 1922.

On examination he was found to be well developed, 6 feet tall, and 233 pounds (105.7 Kg.) in weight. The systolic blood pressure was 190, and the diastolic, 74; the pulse rate was 72. Urinalysis and a blood count gave normal results. The basal metabolic rate was minus 15 and minus 20 on two occasions, but he did not show the typical condition of the skin of a patient with myxedema. A dextrose tolerance test yielded normal values. The fundi and perimetric fields were normal, as was also a localized x-ray picture of the sella turcica. The patient resembled the adult Fröhlich type.

After he had been given desiccated thyroid and the basal metabolic rate had been raised to normal, he felt considerably better and the whole general expression improved so much that one would hardly recognize him as the same person. This improvement, however, was only temporary, and a letter from his wife in October, 1928, revealed that although he was still taking thyroid extract, 1 grain, three times a day, he slept most of the time and continued to have spells of weakness whenever he became excited.

CASE 35.—An oil salesman, aged 39, married, who registered on Oct. 20, 1927, at the age of 9 was kicked on the left side of the head by a small pony, but was not rendered unconscious. At the age of 12 he fell heavily on the pavement, striking his head in the left occipital region, and was semicomatose for three days. Ever since this fall he had been subject to severe, boring, occipital headaches which radiated forward to the left temporal region, causing him to jerk the head backward spasmodically. The headaches occurred once or twice a month, lasted a few hours, and then passed off. In 1920, when the patient was 32, they became more

severe and he consulted an oculist, who gave him glasses that helped him. About the same time he noticed blurring of vision and transitory diplopia of objects near at hand.

At the age of 21, the patient rapidly gained in weight from 120 to 165 pounds (54.4 to 74.8 Kg.), and since then the weight had steadily increased to 200 pounds (90.7 Kg.). At 35, four years before the visit to the clinic, he had influenza, and soon after this noticed sudden attacks of unusual drowsiness when out on a long drive. These increased in severity until, a year before he came to the clinic, they became so marked that he had to stop driving a car. During the past year both the headaches and the drowsiness had improved.

Since the attack of influenza he had also noticed that whenever he was excited or laughed the head suddenly jerked back and he momentarily became limp all over the body. These spells had occurred on attempting to raise a gun to the shoulder to shoot ducks, while attempting to land a fish or while laughing. On sudden exertion, such as starting to run, he suddenly became weak in the knees, but he was always able to catch himself so that he did not fall.

Examination, including a fundoscopic examination, as well as a neurologic study and roentgen examination of the head, gave negative results.

The patient was given deep roentgen therapy to the pituitary area in the hope of empirically helping him, but the day following his return home he died suddenly. Unfortunately, an autopsy was not performed.

CASES OF NARCOLEPSY SHOWING ONLY ATTACKS OF SLEEP

CASE 36.—A married woman, aged 31, came to the clinic on May 24, 1926. At the age of 14, while at school, she noticed that she was having sleepy spells. These gradually became more frequent, and at the time of examination she was having them while talking to friends, while playing bridge, while at the theater or while driving a car. She had practically given up driving a car because of the tendency to go to sleep. Nocturnal rest was disturbed by many dreams, and she frequently talked in her sleep. She was also a somnambulist. Drinking coffee and very active exercise, such as swimming, would forestall an attack of sleepiness. No history of spells of weakness or of influenza was obtained. The father had had a similar trouble, the exact nature of which was not clear.

Physical examination gave essentially negative results. There had been no increase in weight.

CASE 37.—A man, aged 30, a singer, single, who registered on April 22, 1927, had been perfectly well until 9 years of age when suddenly one afternoon he dropped into a sound sleep which lasted about ten minutes. Since then, he had had three or four daily recurrences of sleepiness unless engaged in hard physical labor. Mental activity would not prevent them. The only warning was a slight drowsiness of very short duration, and unless he pinched himself or in some other way caused pain, he would go to sleep. He was unable to go beyond the second year in high school on account of going to sleep in class. He had tried several positions, but was unable to hold any of them for the same reason, so that for the past few years he had been earning his living by singing. He would sometimes go to sleep while walking, but the momentary relaxation of the knees would awaken him. The sleep, while sound, was often accompanied by dreams. He was easily awakened by a light touch, but usually awakened spontaneously after ten minutes of sleep, feeling refreshed. He had never had any spells of weakness. At the age of 12 years he gained weight rapidly, and at the time of examination he weighed 215 pounds (97.5 Kg.); his height was 5 feet, 5 inches (165.1 cm.).

General physical examination, including a roentgen examination of the sella turcica, gave negative results, except for considerable periapical infection. A follow-up letter in March, 1929, showed that there had been practically no change in the patient's symptoms since they started twenty-three years before.

CASE 38.—A blacksmith, aged 31, married, who registered on June 17, 1927, five years before had been seriously ill with influenza and typhoid fever and was delirious for four or five weeks; his memory of this period was a complete blank at the time of examination. Since this illness he had been troubled with frequent sleepy spells which would come on at any time or any place, occurring as frequently as two or three times a day or as infrequently as once a week. The duration of the sleep varied from a few minutes to several hours. He would go to sleep while walking along the street, his pace gradually slowing down until he came to a standstill. He would stand erect with the head bent forward and continue to sleep for a few minutes or until someone awakened him. If he fell asleep while standing still he would not fall, but if while walking, he often stumbled and fell. In many of the sleeping spells he would do unusual things, such as starting up a gasoline engine without knowing that he did so. He frequently talked an unintelligible jargon during the sleeping spells. Three years before, he was forced to give up business on account of this trouble and he had not been able to hold a job since. He dreamed a great deal at night, and his dreams were usually of a troubled and fearful nature which caused him to cry out. He had never had a spell of weakness when laughing or angry.

General physical examination gave negative results, except that emotional reaction in the face was absent. He rarely smiled, and had a stupid, blank expression. He had gained about 40 pounds (18.1 Kg.), and the condition was regarded as a sequel of encephalitis.

CASE 39.—A coal dealer, aged 50, married, who registered on Sept. 27, 1927, for the past three years had had a gradually progressive tendency to lethargy. He would nod for a few minutes, and then go to sleep. The spells would occur as frequently as six or eight times a day and would come on while he was at the office, while carrying on an important business conversation or while playing cards. They had occurred while he was driving a car, and he had narrowly avoided accidents on several occasions. The patient complained of no other symptoms and felt rather foolish about coming to the clinic for such a trivial though troublesome condition.

Physical and neurologic examinations gave negative results. He was 6 feet, 2 inches (188 cm.) tall and weighed 240 pounds (108.8 Kg.). He had had influenza in 1924. Roentgen examination of the head gave negative results.

CASE 40.—A man, aged 32, manager of a wholesale grocery, married, came to the clinic on Aug. 22, 1927. About six months before he had begun to have sleeping spells, which came on suddenly at any time and over which he seemed to have no control. They would last from a few seconds to a minute and would recur in a series of two or three a week with a free interval of from two to three weeks. The spells would come on while he was at a desk reading, or while driving a car. About the same time that the sleepy spells started he began to have morning headaches, three or four times a week. These were aching in character and occurred on awakening, lasting about one hour, but were associated with no other symptoms. He had never had any spells of weakness.

The patient was 5 feet, 9 inches (175.3 cm.) tall and weighed 240 pounds (108.8 Kg.). Between the ages of 22 and 23, his weight had increased from 165 to 225 pounds (74.8 to 102 Kg.). No history of influenza was obtained. Roentgen

examination of the head showed a calcified pineal gland. Examination of the eyes gave negative results. There was no polyuria and no evidence of a definite intracranial lesion.

Case 41.—A farmer, aged 33, married, who registered on March 7, 1928, in 1917 had changed his place of residence and soon after noticed a marked tendency to go to sleep. This had gradually become more severe until at the time of the examination the attacks occurred as frequently as twenty times daily, though they lasted for only a few minutes each time. He would go to sleep while driving a tractor or car, while ploughing or while sitting still in church or at a moving picture, or while talking to friends. He had driven a car off the road on two occasions, but had never had a serious accident. He would frequently get off the tractor to lie down and sleep for fifteen or twenty minutes, waking up spontaneously and feeling refreshed. The sleep seemed to be a normal and natural sleep which he could not resist. The attacks were most apt to occur in the afternoon, and were no more frequent at the time when the patient presented himself for examination than when they started. He had never had any spells of weakness.

Physical examination gave negative results. The patient had had influenza in 1918, after the condition had started.

Case 42.—A truck driver, aged 33, married, who registered on July 19, 1928, during 1924 began to feel tired and was languid and listless while at work, while reading or at almost any occupation that was monotonous. This was especially noticeable while he was at work as a truck driver. There was no particular relationship between the actual amount of work done and the tired feeling, but it seemed rather to be a direct result of the monotony. The condition continued until one year before the visit at the clinic when the tired, languid spells became more severe and he started going to sleep whenever things became dull and monotonous. He had had two automobile accidents occasioned by going to sleep while at the wheel of the truck. The attacks could be warded off by any unusual event or by excitement, such as stopping the car and running up and down the road beside it for a few minutes. If he slept for ten or fifteen minutes he awakened feeling refreshed and could go along for from one to two hours without further trouble. The patient was interested in history, but was unable to read it on account of going to sleep.

He was of a nervous, worrying temperament, vacillating between rather long periods of depression in which he worried unduly about finances, his condition, and his wife and family, even contemplating suicide, and short periods of euphoria in which he felt happy and pleased with the world and his lot in it. It was always during the periods of depression that he had the sleeping spells, never during a period of excitement or euphoria. The euphoric periods were of short duration, lasting only a few hours, while the depressions lasted for several days. Nocturnal sleep was rather restless, and he never had any spells of weakness.

The patient was married and had two children. He was 5 feet, 10 inches (177.8 cm.) tall and weighed 145 pounds (65.8 Kg.). The general physical condition was good, except for some dental and tonsillar sepsis. A history of an attack of influenza in 1918 was obtained.

GENERAL CONSIDERATIONS

True narcolepsy, as a rule, is characterized by two seemingly different kinds of attack; one in which the patient is seized with an irresistible desire to go to sleep, and the other in which the muscles suddenly

relax and give way as a result of some emotional stimulus. The latter attacks have been variously spoken of as the "kataplektische Hemmung" of Henneberg, the "Lachschlag" of Oppenheim, the "Tonusverlust" of Redlich and the "emotional asthenia" of Weech. All of these terms suggest a lack of muscular tone as the basis of the attack, but as some of the phenomena occurring during an attack cannot be explained on this basis alone, Adie preferred to call them cataplectic attacks. The term cataplexy is noncommittal as far as any explanation of the underlying mechanism of the attacks is concerned, and it is to be hoped that, in order to avoid confusion, this name will be adopted generally to designate these attacks; Thrash and Massie, however, in a recent article still confused the term cataplexy with catalepsy, a totally different condition.

There is, however, a certain group of patients who do not have cataplexy, but who have attacks of sleep which are so characteristic of the sleep of true narcolepsy that it seems justifiable to include them under this term, especially the cases in which the attacks have persisted for a long time without the development of any other disease to which they may be attributed. Adie reported one such personal case and two from Goldflam's series; cases 36 to 42, inclusive, here reported fall within this group.

If cases showing brief diurnal attacks of sleep without cataplexy are to be included in the group called narcolepsy, what is one to do about cases which show cataplectic attacks only? Do such cases occur, and if so should they be included among the narcolepsies?

As far as I have been able to determine, the only case of cataplexy without sudden attacks of sleep ever reported was that observed by Gillespie in 1927, but even this patient showed an abnormal drowsiness, especially in the afternoons. Wilson reported a case which he considered to be a larval or incomplete variety of narcolepsy in which the patient suffered from short trancelike periods of immobility, which were broken only by strenuous effort, whenever he fixed his gaze on some definite object as in a day-dream; on excitement or other emotional stimulation also he would become pale and lose control over his legs; however, he did not have attacks of sleep.

Symonds reported the case of a patient who had both kinds of attacks following encephalitis, the attacks of sleep being later replaced by insomnia, while the cataplectic attacks continued. In a case reported by Hilpert, the symptoms began with cataplexy and were followed by attacks of sleep with the eventual disappearance of the cataplectic attacks. In case 19 of this series the cataplectic attacks were the first to appear and were followed in six months by sleeping attacks, but the patient's chief concern in coming to the clinic were the spells of

sudden loss of control of the legs. The cataplectic attacks developed first also in case 23, as the patient was walking downtown after a few days' illness of a mild nature and occurred at first without any especial emotional stimulation; later, attacks of sleep occurred, and finally insomnia became such a troublesome symptom that he came to the clinic to seek relief. The situation was similar in case 32, in which the patient came to the clinic because of spells of weakness, her drowsiness being of secondary concern. Other cases showing both kinds of attacks and in which the cataplectic attacks preceded the attacks of sleep are reported by Willis, by Bostock, by J. K. R. (cited by Symonds) and by Fulton and Bailey.

In the cases in which the two attacks were found in the same patient, the attacks of sleep usually appeared first, lasting from a period of a few months, as in cases 25 and 14, to as long as two years, four and one-half and even six years in cases 9, 12 and 17, respectively, before the onset of cataplectic attacks.

From a study of these cases it seems to me that one need not hesitate to make a diagnosis of narcolepsy in a patient showing attacks of sleep only, provided the attacks are sudden in onset, of short duration and show the other characteristics of the narcoleptic sleep. Adie included three such cases in his report. It may be merely a question of allowing sufficient time to elapse for the development of the cataplectic attacks, but the length of time that may elapse is uncertain; in one case (case 37) the patient had had attacks of sleep every day over a period of twenty-three years without any spells of weakness.

In the majority of cases the two attacks start at approximately the same time (cases 11, 26, 30, 33, 34, etc.), but in only three cases (cases 19, 23 and 32) did the cataplectic attacks precede the attacks of sleep.

CLINICAL MANIFESTATIONS

The Attacks of Sleep.—The term sleep as ordinarily used is very broad, covering in general all grades of somnolence from short transitory attacks of drowsiness to states of such a profound degree that all bodily functions are in partial or complete abeyance. The literature abounds with reports of cases of pathologic sleep, varying all the way from the Fat Boy in Dickens' "Pickwick," who dropped off to sleep at odd moments, to the prolonged sleep of the well known Rip Van Winkle, as well as such cases as those reported by Carlill, Caton, Morton and Janet; the latter reported the case of a patient who slept continuously for five years and was ultimately cured by hypnotism and psychoanalysis. More recently Solomon reported the case of a patient who slept for forty hours and was operated on under hypnosis without any subjective sensation of pain. While such cases are interest-

ing, they are comparatively rare, and the patients usually have some evident psychopathic abnormality in their make-up; as they do not come within the scope of narcolepsy, they need not be discussed further here.

The attacks of sleep in narcolepsy vary greatly both in frequency and in duration in different cases, but are fairly constant for each individual. They may occur as infrequently as four or five times a year (case 22) or as frequently as twenty or thirty times a day. The onset of the attacks may be sudden, coming on "out of a clear sky" without any previous symptom of a disordered condition. Many patients clearly remember their first attack; the patient in case 16 was on his knees weeding the garden when he suddenly fell asleep, and another (case 37), at 9 years of age, suddenly went to sleep one afternoon without apparent cause. In other cases the onset is more gradual, starting as a drowsiness which becomes progressively worse until sudden attacks of sleep develop.

The duration of the attack is also variable; some patients sleep only for very short periods, as did the patient in case 23; he frequently timed himself by his wrist watch and found that he slept only from fifteen to forty-five seconds, or he would go to sleep just as the clock started striking and would awaken before it had finished; other patients sleep for a few minutes, usually from five to fifteen, while still others (case 10) require many hours before they awaken.

The onset of the individual attack of sleep is usually sudden and abrupt, without warning, so that patients may fall asleep before breakfast, even after a good night's rest, or at meals with food half way to their mouths, or while carrying on an important business conversation. This is one of the characteristic observations in narcolepsy and should always be looked for. An aura of short duration occurs in a few cases. In cases 2, 22, 37 and 39, the patients felt drowsy and sleepy for a period of five or ten minutes before actually passing into the sleeping state. In case 15, the patient spoke of a feeling of heaviness over his eyes which always preceded the onset of the attacks of sleep, and the patient in case 4 said that "things seemed to slip far away from her" before an attack. In case 30, the patient stated that a feeling of warmth and heaviness in the head heralded an attack of sleep.

While the attacks of sleep are prone to occur under conditions conducive to normal sleep, such as after a heavy meal, being in a warm room or in an environment of monotony or when fatigued, they are also just as apt to occur under conditions which do not predispose to sleep, and even under conditions in which sleep is very disadvantageous and even dangerous. Patients go to sleep under all conceivable circumstances—while eating, talking or playing bridge, while at the movies or a lecture, while walking, ploughing or driving. One patient

(case 6) went to sleep while outdoors with the temperature 20 degrees below zero, and a school teacher (case 24) used to slip into an adjoining room for a few minutes' nap after he had given the pupils a lesson. Many patients go to sleep while driving a car, even though they fully realize and appreciate the dangers incidental to driving a car while asleep. The reports of automobile accidents in these cases are common, and many patients have been forced to give up driving for this reason. A railway fireman, whose case was reported by Spiller, would go to sleep while hanging onto the grabirons of the engine cab when the train was traveling at a rate of 70 miles per hour.

One patient (case 42) presented an unusual feature in that the attacks of sleep always occurred during periods of depression and never during a period of euphoria. Could the attack be merely an exaggerated form of the depression? That is, since in all depressed states there is usually an associated suppression or inhibition of motor activity, could not the same inhibition extend far enough to produce sleep? If so, in recurring depressions may one not be dealing with another example of internal inhibition as described by Pavlov?

The irresistible nature of the attacks of sleep is shown by the conditions under which they occur. Soldiers under penalty of death would go to sleep while on duty in spite of anything they could do to prevent it. In severe cases the patients shrink from social contacts because of the embarrassment that they suffer by going to sleep while at work, at a bridge party or at any public function. One patient (case 12), even when in a crowd of people, would be forced to lie down and sleep for a few minutes. Another (case 16) would go to sleep whenever he sat down to study in spite of anything he could do, and ultimately had to do his studying while walking around the school campus. Such examples of the patient's inability to resist sleep are common.

A few patients, however, have the ability to resist the attacks wholly or in part, but if they do so they are usually merely postponing them; they have to sleep ultimately. One of Adie's patients said, "If I delay it (the attack of sleep) by moving about I am just packing it up for the future." Working outdoors in the fresh air at hard physical labor was useful in aborting the attacks in three cases (cases 2, 8 and 37). The patient in case 4 was able to avoid a spell of weakness by a conscious effort of the will, but was powerless to alter the attacks of sleep. Another patient (case 30) was sometimes able to continue working through an attack lasting one-half hour, but would have to go to sleep soon after. In case 12, the patient could fight an attack by great mental effort if necessary, but it was much less exertion to

go to sleep for a few minutes. If the patient referred to in case 27 wished to go to a party, she would sleep for two or three hours before in an effort to keep awake at the required time. One patient (case 15) could occasionally, but not always, stall off an attack. In case 17, the patient was able to ward off an attack for a while by walking around, but it would eventually overpower him. Another patient (case 21) could sometimes avoid an attack by vigorous exercise or by washing his face in cold water. In spite of the irresistible nature of the attacks of sleep, a characteristic observation is that a very slight stimulus from some outside source will be sufficient to awaken the patient and bring him thoroughly out of the attack. The attacks are invariably worse after a night of restless sleep, but a good night's rest will not prevent their occurrence the following day.

Some patients continue to perform automatic or reflex movements after going to sleep; one patient (case 15) would continue to walk behind a team while ploughing until he reached the end of the field where the horses stopped; he would then awaken after having walked a distance of perhaps 60 yards while asleep. If another patient (case 6) fell asleep while talking or writing, he would continue to perform these acts automatically, speaking an incoherent and disconnected jargon and making meaningless marks on the paper. In case 38, the patient might go to sleep as he walked along the street, his pace gradually becoming slower until he stopped or stumbled and fell. He also performed some unusual acts during these spells, and on one occasion started a gasoline engine without any knowledge, or at least any remembrance, of having done so.

Most of the attacks of sleep are described as attacks of normal sleep, but the more carefully one examines them the more fully one realizes the many differences that exist between the sleep of the narcoleptic patient and normal sleep. The sudden onset, the irresistible nature in spite of a good night's rest and the short duration of the attacks which are so characteristic of narcolepsy are not found in normal sleep. As a rule, the sleep is extremely light, and the slightest touch or stimulus from some outside source is often sufficient to awaken the patient. No matter how short the period of sleep may be, the patients invariably awaken feeling refreshed, mentally clear and alert, and with all the peculiar feelings of drowsiness that may have been present before the attack completely dissipated. Many of the less severe cataplectic attacks in which the patient becomes motionless with the muscles relaxed and the eyes closed and is unable to speak or answer questions appear to be attacks of sleep and are frequently described as such, but the patients are invariably conscious during the whole attack.

The fact that some of the patients are dimly conscious of what is going on but totally unable to move a muscle to help themselves strongly argues against the attacks being of the nature of normal sleep. The occurrence of dreams during the diurnal attacks of sleep and the nocturnal restlessness seen in these cases will be discussed later.

Cataplectic Attacks.—Cataplectic attacks are characterized by a sudden loss of muscle tonus in the patient's skeletal musculature so that the affected muscles become weak and powerless, allowing him to sink to the ground totally helpless; after a brief period, usually from a few seconds to a few minutes, the strength and power return and the patient is able to get up unaided, feeling none the worse for his experience.

Attacks similar to these are characteristic of narcolepsy and occurred in thirty-five of the forty-two cases. The attacks, like the attacks of sleep, vary widely both in degree and in the muscles involved. Sometimes the patient experiences no more than a fleeting weakness in the knees if standing up or in the dorsal spine if he is in the sitting posture, while others may have a generalized weakness affecting all the skeletal muscles and lasting for a period of five or ten minutes.

Clinically, these attacks occur as a loss of muscle tonus, which is recognized as a definite entity but which cannot be clearly defined or differentiated; there is also a diminution or loss of volitional power. These two phenomena are closely related, and it is hard to separate one from the other, but both occur in narcolepsy; in the one, an arm or leg suddenly loses its muscle tonus and becomes flaccid, weak and powerless to perform a definite act, and in the other a patient is suddenly inhibited from doing something he had willed to do. One patient (case 31) would suddenly become weak whenever he attempted to throw a stone; another (case 35) noted muscular weakness on sudden exertion or whenever he started to run, and another (case 23) on attempting to shake hands would often find himself unable to do so, his arm having fallen limply to his side. In some of these cases it is hard to determine what precipitated the spell of weakness, whether it was the mere volition to throw a stone, to run or to shake hands, or whether it was the emotion that engendered a desire in the patient to do those things. However, in some cases, it seems more logical to attribute the spells of weakness to the mere act of volition itself. The patient in case 23 is of interest in this connection in that on two or three occasions while in bed, just as he was about to go to sleep, he suddenly became faint, weak and powerless to move and thought he was going to die. He was able to call to his mother, and when she came and touched him he immediately came out of the attack. Here, all voluntary motion except that connected with speech seemed to be

in complete abeyance and some outside stimulation was necessary to overcome it. The patient in case 18 had a similar condition in that at night he often had the feeling that someone was crawling in at the window; he would tremble and shake all over and be totally powerless to move a muscle or even to speak, but if his wife, recognizing his condition, would touch him, he would immediately be himself again. Another patient (case 9) had had several similar experiences at night, sometimes feeling that people were stealing his blankets, and was totally unable to make the slightest effort to save them; at other times when he felt that there was some one in the room, he was able to reassure himself by getting up and looking around. It is curious that several of these patients were impressed with the same fear, namely, that some one, usually a stranger, was in the room with them.

The loss of muscle tonus may be localized, as in the case of the patient (case 26) who complained of "muscle jerkings" during which he suddenly lost power in certain groups of muscles so that his head would fall forward, his arm give way or his knees become weak; other patients, as in case 10, would shake and tremble over the whole body. In still other cases the weakness would start locally and become generalized, sometimes suddenly and sometimes gradually. In case 24, the patient stated that on attempting to throw something he was seized with a feeling of weakness and faintness which started in the legs and passed as a wave over the whole body. Another patient (case 2) stated that his first attack, which came on while working, consisted of a gradually generalized muscular weakness, whereas the patient in case 11 said that he sank slowly to the floor while dancing with his daughter.

The association of various degrees of muscular movement with the attack is by no means an uncommon observation and has been reported previously. The patient in case 26 reported that his muscles twitched and jerked after he had fallen, while other patients merely shook and trembled during the attack. The occurrence of a fine tremor of the muscles of the face and hands was noted in one patient (case 24) who had an attack while being examined. In another (case 17), who also was examined during an attack, the lower jaw trembled, the facial muscles twitched and the deep tendon reflexes were greatly diminished or abolished. The patient in case 31 had short, quick, jerking movements of the shoulders and elbows, which at first she was able to pass off as laughing spells but which later became more severe and caused her to drop things while at the table. Some patients lose speech entirely during an attack, while others mumble inarticulate words or make grunting noises. Blurring of vision with diplopia was observed in several cases (cases 2, 9, 32 and 34), owing probably to

a transient imbalance of the extra-ocular muscles resulting from the patchy and uneven distribution of the loss of muscle tonus.

A striking and characteristic feature of all these attacks of weakness is the maintenance of perfect consciousness throughout, in spite of the severity of the attack. As a rule, consciousness is in no way impaired, and in only two instances was there any departure from this rule; one patient (case 18) said that he was only dimly conscious of what was going on, and another (case 24) said that his mind became cloudy during an attack.

The precipitating cause of these sudden spells of weakness is usually some emotional stimulus, such as mirth, anger, fear, surprise, joy or amusement. The act of laughing seems to be the most frequent factor in provoking the attacks, and some patients learn to prevent them by restraining their mirth and refusing to laugh. Illustrative examples of conditions under which spells of weakness occur have been given elsewhere and hardly need be repeated, except to mention a few additional ones. The patients in cases 7, 13 and 35 were sportsmen and were prone to have attacks just as they raised the guns to their shoulders preparatory to shooting; or an attack would occur on hooking a fish, and one patient (case 13) was unfortunate enough to lose his fishing rod on such an occasion. Sudden meeting with friends while walking along the street, or the sight of dogs, snakes or anything unusual are common causes of spells of weakness. That not all emotions will precipitate an attack is shown by the patient (case 17) who would have an attack just as he reached the climax of a funny story that he was telling or at an exciting play in a card game, but who a year previously had cursed a man in a fit of anger for an hour without bringing on an attack; yet he had an attack during examination when he attempted to express gratitude for a cigaret. When the patient in case 26 found that a shiny object lying in the middle of the road was a gold watch he immediately fell in a heap.

A few patients have, to a slight extent, the ability to resist an attack of weakness. One patient (case 24) had an attack while in the office when he attempted to throw a towel, but a second attack failed because he said his mind was on it, maintaining that by the state of his own mind he could fortify himself against the spells if he had sufficient warning. The patient in case 4 could control the spells of weakness by a conscious effort of the will, but could not prevent the attacks of sleep. Another (case 21) could sometimes prevent an attack by tensing the muscles of the leg or tongue, but this took great mental effort.

The occurrence of somatic sensations with the attacks has been mentioned, but they are rather infrequent. The patient in case 4

complained of a dry mouth; the one in case 13 became pale and had vertigo. In case 9, the patient complained of his hands and feet "going to sleep," while the patient in case 31 trembled inwardly and another, case 20, complained of a gone and sinking feeling.

Transitional Attacks.—While typical examples of the two types of attacks previously described are distinct and dissimilar, there is a group of intermediary attacks in which it is hard to differentiate one from the other; for example, when one patient (case 3) had an attack in which his head fell forward onto his chest, his eyes closed and he was unable to talk, his mother thought that he was asleep or unconscious until he assured her afterward that he was perfectly conscious throughout the attack. Numerous other similar cases appear to the onlooker to resemble normal sleep, while still other cases show equally interesting transitional forms of attack.

Gélineau's original patient would suddenly go to sleep under strong emotional stimulation, and one patient in my series (case 22) noticed that laughing and excitement tended to bring on an attack of sleep. Another case reported by Kahler showed the same relationship, and Miller cited the case of a young woman in whom strong emotion produced a state of sleepiness.

The patient in case 6 noticed that if he tried to fight an attack of sleep he would become trembly and shaky over the whole body, and that the knees would give way and he would fall to the ground. A similar thing happened to the patient in case 10 and in two cases reported by Wilson. Here the resistance of an attack of sleep resulted in a cataplectic attack. One patient (case 21) noticed that a cataplectic attack was sometimes followed immediately by or passed into an attack of sleep.

In one patient (case 23) the cataplectic attacks developed spontaneously, as do most of the attacks of sleep, without any particular reference to emotional stimuli, although they became associated later. This patient also noticed attacks of weakness in which he was unable to move a muscle, which came on just as he was about to drop off to sleep after he had gone to bed at night. In such a case it seems probable that the inhibition that had intercepted the centripetal sensory stimuli preparatory to the sleep state had also rapidly spread to involve the proprioceptive stimuli arising from the musculature, thus producing a state of flaccid immobility.

Other Phenomena.—As previously pointed out, the attacks of sleep exhibit so many peculiar characteristics that one may justly question their identification with sleep as it is observed in a normal person. There are, however, sufficient similarities to make it apparent that a

close relationship does exist between them, so that the causative mechanism of one may have a bearing on the production of the other. The clinical criteria for the diagnosis of sleep are so variable and uncertain that a definition of the sleeping state is by no means easy. Johnson assigned the word "sleep" to a condition characterized by a "high degree of muscular relaxation accompanied by an enormous increase in the magnitude of the stimuli necessary to arouse a specific response." This definition fails to account for the complete loss during sleep of critical reactivity of the individual by which sensations are normally analyzed and interpreted (i. e., consciousness). In the condition known as myoplegia or periodic family paralysis, there is such a complete and total relaxation of the musculature that any amount of stimulation will fail to produce a response although the patient is not asleep and consciousness is undisturbed.

The work of Richter on electric resistance of the skin may prove a valuable aid in the diagnosis of sleep, although this method is criticized by Johnson, Swan and Weigand. Richter has shown that normal subjects gave fairly constant results, while the results in patients with catatonic and schizophrenic tendencies showed wide variations. A later study of patients showing symptoms of narcolepsy showed the following variations: (1) that the average daily resistance of the skin was much higher than in normal persons, frequently being as high in the waking state of the narcoleptic person as during the sleeping state of normal persons; (2) that there were wide fluctuations not only from day to day, but from moment to moment, and (3) that the resistance increased rapidly to a high level when a patient was left undisturbed in a quiet place for a few minutes, whereas in normal subjects it tends to decrease. While these observations do not aid materially in clarifying the underlying problems of narcolepsy, they do show that some fundamental psychomotor change has taken place in these persons.

Another phenomenon that seems important is the relationship of the deep reflexes during sleep and during a cataplectic attack. According to Tuttle, Lombard, in 1887, was the first to discover that the knee reflexes were either greatly diminished or abolished during sleep. Since that time many similar observations, including the presence of a positive Babinski response, have been made by Collier, Kleitman and others. Wilson observed abolition of the knee reflexes with a positive extensor response in his patient, and a loss of knee reflexes during an attack was observed in the patient in case 17 of my series; Babinski's test was not made. Worster-Drought and Purves-Stewart reported similar observations. The mechanism of similar reflex changes in both

cataplectic attacks and normal sleep is not clear, but the changes at least suggest a common disturbance of the pyramidal system.

Mauthner considered sleep to be due to a center in the region of the oculomotor nuclei of the brain stem which had the power of blocking off incoming sensory impulses. Haberman and, later, Kleitman, basing their opinions on the occurrence of a positive Babinski response during sleep, which could be overcome and negatived by repeated stimulation, regarded it as being due to an increased synaptic resistance from a functional block of the pyramidal system. Kleitman, reviving the teachings of Hughlings Jackson on the presence of various levels in the central nervous system, attributed sleep "to fatigue of the highest centers of consciousness, and dreaming to the persistence of activity of the lower centers."

While in general, definitions of sleep agree to a greater or less extent, the one proposed by Pieron seems the most satisfactory. According to him, sleep consists of a suspension of all the sensorimotor activities which bring a living organism into relation with its environment. The mechanism by which this is brought about is not definite, but receives a plausible explanation in Pavlov's work that will be discussed later.

The variability of the clinical phenomena which these cases present, with the numerous transitional forms of attack which may occur, forces me to the opinion that what one sees clinically is the variable symptom-complex of a condition the etiology of which is some inhibitory mechanism of the cerebral function.

ASSOCIATED CONDITIONS

Restless Nocturnal Sleep with Dreams.—In a large proportion of cases, the nocturnal sleep of the narcoleptic person is restless and disturbed by dreams, in spite of reports to the contrary. Wendoveric stated that the attacks of sleep of narcolepsy were distinguishable from normal sleep by the occurrence of "grelle Traüme," but this assertion does not seem wholly warranted, and Adie took exception to it, saying that vivid dreams occur frequently in normal persons. Wilson, however, stated that "in the majority of instances the nocturnal sleep of the narcoleptic is normal," a statement which, according to the cases here reported, should not pass unchallenged.

No mention was made of nocturnal sleep in seventeen of the forty-two cases here presented. Of the remaining twenty-five cases, specific mention was made of the occurrence of restless nocturnal sleep in eighteen, or 72 per cent. In only seven was there a record of good

nocturnal sleep. The restlessness may vary from frequent turning and awakening to vivid dreams of a wild and exciting nature. A few illustrative examples will show the nature of this extreme restlessness.

CASE 4.—Running parallel with the diurnal drowsiness, the patient developed nocturnal restlessness during which she kicked, tossed and thrashed about in bed, talking and laughing to such an extent that no one could sleep in the same room with her. Her dreams were usually one of three types: (1) fear of the death of her child, (2) problems, in which she was continually working out an everlasting experiment and (3) mirth.

CASE 7.—The patient had "terrific nightmares" in which he would toss and twist about in bed, crying, talking and, in general, carrying on such a fuss that no one would sleep with him. He was always more alert at night than in the daytime, and for this reason he usually did not go to bed until 3 or 4 a. m.

CASE 17.—The patient had been a restless sleeper for twenty years, tossing and twisting about in bed, screaming, crying, talking and cursing so loudly that neighbors could hear him. In 1924, the dreams became "terrible and horrible," and he was usually being chased by snakes and other animals; these terrifying and unpleasant experiences would often wake him up, and the emotion of fear persisting over into the waking state would precipitate a spell of weakness.

CASE 24.—About four hours after retiring, the patient began to have dreams of a wild and exciting nature which caused him to toss about in bed, to call out and to talk. These would persist until morning.

CASE 18.—The feeling that some one was trying to crawl in at the window would produce an attack of weakness and trembling in this patient, but a slight touch from his wife would suffice to break the spell.

CASES 2, 8 and 27.—The patients occasionally dreamed during short diurnal attacks of sleep.

From such cases it is evident that the restlessness of the nocturnal sleep must have some bearing on the subject of narcolepsy. Why these people should have such clear and vivid dreams is hard to determine, but the dreams must surely be an evidence of some form of cerebral activity indicating that the disease process, whatever it may be exerts its influence not only diurnally but also nocturnally.

Obesity.[1]—Obesity is commonly associated with narcolepsy. Eighteen of the patients of my series reported a definite gain in weight, varying in amounts up to 100 pounds (45.4 Kg.) (case 32), with an average of 27 pounds (12.2 Kg.). The gain in weight was usually coincident with or preceded the onset of the narcoleptic symptoms.

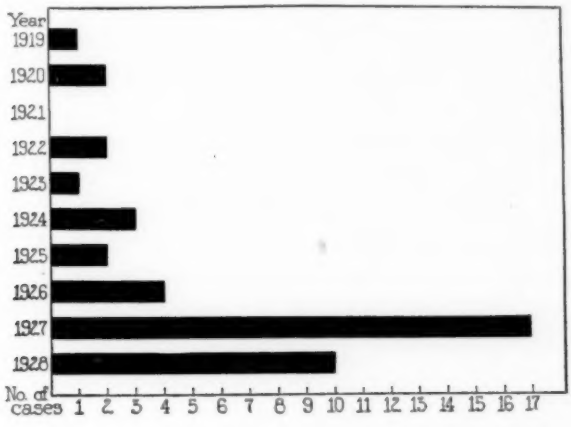
Of the eight female patients, six were definitely obese, the average weight being 202 pounds (91.6 Kg.), averaging 66 pounds (29.9 Kg.) overweight for the height and age. Two were underweight, but the average for all the females was 45 pounds (20.4 Kg.) overweight.

1. Comparisons of age, height and weight were made with the tables supplied by the Metropolitan Life Insurance Company.

Of the males, twenty-nine were found to average 30 pounds (13.6 Kg.) overweight, while only four were underweight, the average for all the males being 24 pounds (10.9 Kg.) overweight.

ETIOLOGY

Frequency.—The rarity of this condition has been mentioned previously and is further emphasized by the absence of any mention of narcolepsy in the third edition of Osler's "Modern Medicine," while "Oxford Medicine" (1920) curtly dispenses with it in one brief sentence, saying that "Narcolepsy is a state of profound sleep into which hysterical subjects are liable to fall suddenly."



Increase in the number of cases between 1919 and 1928, inclusive.

Eleven of the patients in my cases registered at the clinic during the years from 1919 to 1925, inclusive, averaging almost 2 per year. In 1926, 4 cases were registered, while in 1927 there were 17 and in 1928, 10 (chart). Why there should have been such a sudden increase in the incidence of this condition in 1927 is not clear. The 42 cases represent a total registration of 361,602 patients, or an average of 1 case of narcolepsy in every 8,610 patients.

Sex.—Of the forty-two cases reported, thirty-four were males and only eight females, a proportion of approximately four to one in favor of the males. Wilson reported a somewhat higher ratio, there being thirty-seven males to seven females among the cases that he was able to collect from personal experience and from the literature. Why this discrepancy should occur in the frequency of the condition in the two sexes is difficult to understand and hard to explain. It has even been said that true narcolepsy is limited to the male sex, but this is

untrue, as shown from this series as well as from those reported by Adie, Wilson, Weech and Redlich.

Age and Onset.—The age at onset in the males varied from 9 to 46 years, and averaged 25 years. The duration of symptoms before coming to the clinic, in males, varied from six months to twenty-one years, with an average of five years.

The age at onset in the females varied from 8 to 37 years and averaged 19 years, six years earlier than in the males. The duration of symptoms in females before coming to the clinic varied from four to seventeen years, and averaged ten years. The average age at onset in both males and females was 24 years, a figure agreeing closely with 23½ years given by Adie. It is interesting to note that the average duration of symptoms in the males before coming to the clinic was five years as compared to ten years in the females. That is, the males sought medical relief twice as soon as the females. This is probably due to the fact that females, being engaged in less active work, suffer less inconvenience from the attacks than do the males.

Personal and Family History.—As a rule, there is nothing in the family history to denote any neuropathic or degenerative hereditary factors, as McCarthy and Blodgett would lead one to believe. Nor has one any evidence to show that syphilis plays a significant part in the etiology of the condition, since only one patient (case 4) in the series showed a positive Wassermann reaction.

The father of one patient (case 36) was said to have had a similar condition, but its exact nature was uncertain. The mother of one patient (case 16) had had two nervous breakdowns, and a maternal aunt was a patient in a state hospital for the mentally defective. In case 32, the patient came from a family of exceptionally obese people, but she was the only one of the family who suffered from ill health. Headaches and migraine did not seem to occur any more frequently in this series than in any other group of patients. Epistaxis has been observed in cases of typical narcolepsy by Wilson, and Weech recently reported a case in a child, but no instance of it was found in this group.

Trauma may be mentioned as of possible etiologic importance in one patient (case 35) who was kicked on the left side of the head at the age of 9. At 12 he fell on a sidewalk, bumping his head in the left occipital region, and was semicomatose for three days. Following this he suffered from headaches and visual disturbance until he was about 35 years of age, when the narcoleptic symptoms began. Trauma has also been noted in cases reported by Wilson, Redlick and others.

Cases have been reported by Carlill, Meyers and Morton in which the narcolepsy seemed to be on an hysterical basis and was either

cured or helped by psychoanalysis, but careful study of these cases shows that they do not fall within the category of true narcolepsy. The one patient of my series whose symptoms could possibly be explained on a psychologic basis was the patient in case 20, out of which any Freudian could make a very exhaustive and interesting, if not illuminating, discussion.

Of nine cases reported by Redlich in which a complete blood count was done, six showed lymphocytosis. In my group complete blood counts were done in only seven cases, the lymphocytes varying from 23.5 to 38 per cent, with an average of 30 per cent, a figure slightly above normal, the significance of which is unknown.

Mentally, my patients were normal in all instances, being capable of earning a living for themselves and their families. They come from all classes of society, including students, teachers, clerks, insurance agents, farmers and housewives. The fact that some of the patients became incapacitated and had to change occupation to suit the condition speaks for the ability of these people to make adjustments to existing conditions.

COURSE, PROGNOSIS AND TREATMENT

From a review of my cases and those in the literature, one may say that narcolepsy is essentially a chronic condition, running a fairly constant course and showing a characteristic and stereotyped symptomatology. The symptoms usually reach their maximum intensity soon after they start, and remain at that level for many years. If the patients are not totally incapacitated, they can usually find some form of work to which they can adapt themselves in spite of the handicap. Remissions do occur in a limited number of cases. One patient (case 30) showed temporary improvement for three months during the winter, and another (case 25) was much worse during the first year of the disease than at any other time. The patient in case 23 had so many attacks during the first year of the disease that he hardly dared to venture outside alone, but he later improved sufficiently to resume work as a school teacher.

The length of time over which symptoms may persist is at present unknown; Wilson and Goldflam each reported a case in which the patient had had symptoms for seventeen years, and two patients (cases 8 and 32) had had symptoms for fifteen years each. The prognosis, then, is uncertain, but is essentially that of a chronic disease.

Treatment is likewise unsatisfactory, being purely symptomatic, since there is no specific drug known for this condition. Bromides, phenobarbital and other anti-epileptic forms of treatment have been tried, with disappointing results. Frequent periods of rest during the day tend

to lessen the frequency of the attacks, and two of the patients in the cases reported made this a useful therapeutic measure if they wanted to go out for an evening's entertainment and avoid the embarrassment of frequent narcoleptic attacks. Bostock found a mixture of caffeine, strychnine and opium useful in reducing the frequency of attacks; Redlich recommended frequent periods of rest, nux vomica and desiccated thyroid extract. One patient (case 32) who had a basal metabolic rate of minus 17 was given thyroid extract until the rate became normal, but there was no improvement in the symptoms; another patient (case 34), whose basal metabolic rate was minus 15 and minus 20 on two occasions, improved considerably for a while after the administration of thyroid extract. It was also thought to have been beneficial in cases 12 and 26. In one case (case 27) sodium cacodylate was given intramuscularly, and temporary benefit was noted. Deep roentgen therapy was used in a few cases in which it was thought that there might be a neoplastic lesion of the base of the brain, but in only one case (case 33) was there any beneficial effect.

NOTE.—Since this paper was submitted for publication, Dr. L. E. Daniels of the Mayo Clinic published in the *Proceedings of the Staff Meetings of the Mayo Clinic* (**5:** 399, 1930) a suggestion regarding the use of ephedrine in cases of narcolepsy. In five cases it gave complete and prompt relief.

I used ephedrine with good results in one case in which as many as thirty attacks of sleep occurred daily; I was able to control the attacks with the use of 50 mg. of ephedrine three times a day, thereby making it possible for the patient to carry on his occupation as a salesman. This case was not included in the forty-two reported in this article.

PATHOLOGY

The relationship of narcolepsy to organic diseases of the brain is undetermined, and up to the present there has been no published report of an autopsy in a case of narcolepsy. Readers are requested to bear this in mind, and if possible to obtain one in the hope of clearing up some of the problems connected with this strange symptom-complex.

Somnolence associated with tumors of the brain has been noted for a long time, but Hercouët, in 1905, in a careful analysis of sleep in reference to tumors of the brain, concluded that there was no localizing value in somnolence alone, but that it occurred most frequently in tumors at the base of the brain. The somnolence of the narcoleptic patient, however, is different from that associated with organic disease of the brain, and one must not assume that the pathologic changes are the same in both cases. Intracranial growths are usually sufficiently

large to produce not only direct involvement, but symptoms due to toxins, edema and pressure, all of which may play a part in the production of somnolence.

Baruk, Bertrand and Souques reported the case of a woman, aged 37, who complained of headache, visual disturbances and sleepiness for six months. For two or three months she had had frequent attacks of sleep of short duration from which she awakened easily. Autopsy showed a small tumor in the region of the tuber cinereum and infundibulum of the third ventricle.

Lucksch reported a case of multiple embolic abscesses in the walls of the third ventricle, and Pette reported a thrombotic lesion in the same region, both patients showing a lethargic condition similar to that observed in epidemic encephalitis; in each case the lesion was located in the region of the nucleus of the third nerve, the aqueduct of Sylvius and the adjacent part of the thalamus. Pette thought that this region probably contained the center controlling sleep itself.

McKendree and Feinier, reviewing 100 cases of neoplasm of the brain with respect to somnolence, found it present in 32 cases, but concluded that somnolence as such was of no value as a localizing sign in intracranial lesions.

The hypothalamic syndrome or symptoms due to involvement of the infundibulum are characterized by varying degrees of hypersomnia, polyuria, adiposity and genital dystrophy. Any one, or all, or any combination of these symptoms may occur, depending on the specific region involved. Fulton and Bailey believed from a review of the subject that it is wrong to speak of a "sleep center" as such, and that the region responsible for disturbances of the rhythm of sleep, but not necessarily for hypersomnia, lies farthest back in the region of the gray matter near the anterior extremity of the aqueduct of Sylvius. Von Economo, in a study of epidemic encephalitis, came to the same conclusion; namely, that lesions around the third ventricle were responsible for the change in the rhythm of sleep so characteristic of that disease.

The frequent occurrence of adiposity in many cases may lead one to the site of the pathologic changes present. Experimental production of adiposity by lesions of the tuber cinereum has been accomplished in dogs by Bailey and Bremer and in rats by Smith. In spite of the care taken to limit the amount of damage done in these experiments, polyuria, genital dystrophy and glycosuria occurred frequently, as they did also in experiments by Camus and Roussy, and were probably due to involvement of the infundibulum.

The patient in case 32 not only came from a family characterized by obesity, but gained 100 pounds (45.4 Kg.) during the three years

in which the narcoleptic symptoms were present. Besides an increased thirst and appetite with a rapid gain of 40 pounds (18.1 Kg.), the patient in case 33 had undergone a change in character. Another patient (case 34) had gained 70 pounds (31.8 Kg.), and had become mentally dull and impotent, with frequent headaches. Physically, he presented the adult Fröhlich type with a low basal metabolic rate, and while it was generally thought that the patient had a lesion involving the hypothalamic region, it was not believed that exploration was justifiable. The patient in case 35 had suffered from trauma to the head, followed by headache and a rapid gain in weight. The possibility of an organic lesion at the base prompted the use of deep roentgen therapy to the pituitary region, but the day following his return home he died suddenly. Autopsy was not performed, so that one can only speculate as to the cause of death, but one might make the supposition of a hemorrhage into a slowly growing neoplasm softened by roentgen therapy.

THE RELATION OF NARCOLEPSY TO OTHER CLINICAL CONDITIONS

Periodic Family Paralysis or Myoplegia.—Wendoveric and, more recently, Wilson called attention to the striking similarity between the cataplectic attacks of narcolepsy and those of periodic family paralysis. Both consist of a sudden widespread paralysis of the skeletal musculature of the flaccid type with abolition of all deep reflexes. The cataplectic attacks, however, are of shorter duration and of more sudden onset than the paralysis of myoplegia, but the sudden and complete loss of muscular tone in the two conditions argues for a common etiology.

Catalepsy.—This indefinite term covers a multitude of conditions, but is usually meant to convey an idea of muscular immobility with either partial or no clouding of consciousness. Such a condition is often spoken of as a trance, and has long been associated with such phrases as "paralyzed with fear," "speechless with surprise," etc. Patients in this condition often exhibit the "cerea flexibilitas" so characteristic of catatonia. In my cases are several examples in which the patient suddenly passes into such a trancelike state, perfectly aware of his surroundings, but utterly helpless and unable to move or even speak in some cases.

Epilepsy.—The clinical pictures produced by epilepsy in all its various forms are numerous and familiar to every doctor. They vary from the so-called epileptic equivalent through petit mal attacks to severe convulsive seizures. The relationship of these attacks to those seen in narcolepsy is not clear or definite. Curschmann and

Prange even advocated the abandonment of the term narcolepsy, because they said that it implies a relationship to epilepsy that does not exist. Adie, likewise, was emphatic in saying that there is no connection between the two conditions. Kahler, Goldflam and others were equally certain of the differences between them. Wilson, on the other hand, saw a great many similarities between the two conditions, and pleaded for a little less absolutism and quibbling over the terms used to denote conditions the etiology of which still remains uncertain. Wilson aptly asked for a definition of epilepsy before one can say that the two conditions have nothing in common, and maintained that "any alleged contrast between the narcolepsies and the epilepsies is merely a contrast between symptoms and leaves untouched the problem of whether or not there is a similar mechanism of production behind each."

Clinically, the two conditions have attacks that have much in common. The periodic recurrence of attacks of sudden onset, either with or without an aura, are common to both. The muscular jerkings and involuntary movements, either local or generalized, seen in many of the cataplectic attacks closely resemble those seen in mild forms of epilepsy. These have been discussed elsewhere in this paper. One of the essential characteristics of all epileptic attacks is loss of consciousness, while the retention of consciousness is equally characteristic of the cataplectic attacks of narcolepsy, but in two cases (cases 18 and 24) of this series the patients, curiously enough, complained of a diminution of consciousness during the spells of weakness. One patient (case 5) had peculiar attacks in which he would do certain things without really knowing what he was doing, there being apparently a momentary loss of consciousness, but since they were associated with both spells of weakness and attacks of sleep it became difficult to separate one from the other. Unfortunately, this patient could not be traced, and I do not know what became of him. In case 18, the patient had peculiar attacks in which he became dyspneic, a glimmer appeared before his eyes, his body became weak and limp and he would have to lean up against something for support. He also had a fine tremor of the hands and face with slight convulsive movements of the arms and legs, and was only dimly conscious of what was going on. Are such attacks to be regarded as epileptic or narcoleptic? In case 3, the mother always thought that the patient was unconscious during an attack until he assured her to the contrary. These and other cases show the marked similarity between some of the attacks and those of epilepsy, making a differentiation between them a little more difficult than some would lead one to believe.

The association of both epilepsy and narcolepsy in the same person has been noted by several observers. Gowers reported the case of

a girl, aged 14, with a family history of epilepsy, who had had characteristic convulsive attacks for four months and had also had "attacks of sudden pure sleep." On the administration of bromide, the epileptic seizures disappeared, but the attacks of sleep remained. Worster-Drought also reported the case of a man, aged 25, who had both forms of attacks. One of Goldflam's patients suffered from narcolepsy for six years and later developed typical epilepsy with convulsive seizures. Wilson reported a case showing a transition from epileptic to narcoleptic attacks. Zador reported the case of a woman, aged 32, who showed both kinds of attacks and whose mother suffered from atypical epilepsy; while at school the patient suffered from attacks of vertigo, unconsciousness and migraine, which with the onset of the menses were replaced by spells of nausea and vomiting; later, both types of narcoleptic attack occurred; the attacks usually lasted about two or three minutes and followed excitement or fear; they were characterized by a sense of nausea, followed by loss of muscular tone and sleep; during the early part of the attack she could not be aroused, and after it was over she had no recollection of what had happened. These attacks were suggestive of epilepsy, but their occurrence under emotion argued for narcolepsy. From this case Zador concluded that there was a gradual transition from the early attacks of vertigo and unconsciousness through migraine to narcolepsy and epilepsy, the same mechanism operating throughout, and also that epilepsy and narcolepsy had so much in common that it was not possible to draw a distinct line between them.

Stewart reported an interesting and illuminating case in this connection. In a child who was subject to epileptic seizures, cataplectic attacks developed on the first taste of food, which Stewart assumed acted as an emotional stimulus. Later, both cataplectic and epileptic attacks were replaced by periodic attacks of sleep. Examination during one of the cataplectic attacks showed that the knee reflexes were absent, with an absent or indefinite extensor plantar response.

In attempting to show either the similarities or the differences in the two conditions, one should keep in mind the variability of the reflex changes, with or without a Babinski response, that occur in epilepsy, and the frequent observations of the loss of deep tendon reflexes in narcolepsy with the extensor response in the case reported by Wilson. These changes warrant at least more than superficial consideration.

The failure of certain patients with narcolepsy to respond to the medication prescribed for epilepsy is not conclusive evidence for disproving the similarity of the two conditions; the same is true of the so-called "epileptic personality."

Although narcolepsy and epilepsy have much in common, it must not be assumed that they are identical; narcolepsy is of comparatively recent observation and of increasing frequency as compared with epilepsy, which has been known since the time of Hippocrates.

From such clinical observations one may argue both for and against the relationship of the two conditions, but to say that there is no connection between the two is an assumption entirely unwarranted. I agree with Wilson, who said that "the correct attitude is to suspend judgment and await further contributions to our knowledge."

THEORETICAL CONSIDERATIONS

Sleep is such a natural occurrence in every-day life that one pays little attention to the physiologic principles underlying its production. Its opposite, insomnia, however, does have the effect of stimulating inquiry, and persons afflicted with this distressing symptom are eager in their solicitations for help from the physician, who, unfortunately, in regard to the physiologic and pathologic problems involved is apt to be almost as ignorant as the person who seeks relief. One of the reasons for this ignorance is the lack of experimental investigation, which is difficult because of the use of animals of different species and ages, and because of the different methods used to produce and maintain insomnia. Kleitman, using puppies, Kleitman and Lee, using human beings, Bast and Bloemendal, and Leake, Grab and Senn, employing rabbits, attacked the problem of sleep from the negative aspect; that is, they studied experimentally produced insomnia. To do this, some means of muscular exercise must be employed to keep the animals awake, and Kleitman, one of the most recent workers, emphasized the fact that it is impossible to divorce experimental insomnia from muscular activity; for this reason he admitted that even his own results are of doubtful value. Pavlov, on the other hand, quite accidentally, began the study of sleep from the positive side, owing to the fact that dogs persisted in going to sleep during the course of experimental studies on conditioned reflexes.

The various theories that have been advanced from time to time to explain the mechanism of normal sleep are briefly and fully reviewed by Callender, Kleitman and Jones, and need not be considered here since they have all been more or less displaced by the worthy investigations of Pavlov. A brief review of this work as it relates to normal sleep may be of value in attempting to understand the underlying mechanisms of narcolepsy.

Pavlov's work on the nervous activity of dogs was carried out on a purely physiologic basis; psychologic conceptions or words were never used. The highest nervous activity of an organism consists in the

synthesis and analysis of the stimuli which reach the cerebral cortex both from without and from within the organism. The basis of all nervous activity depends on the so-called reflexes, which are connections established between definite outside agencies and corresponding definite activities of the organism. Pavlov, who considered all acts as reflex in character, distinguished two kinds: (1) the simple, physiologic reflex, inborn, instinctive and unalterable, which he called the unconditioned reflex, and (2) the more complicated reactions usually called psychic, which are acquired and are formed only under certain conditions, which he called the conditioned reflex. This can be more fully understood by a few examples from his work.

If some external stimulus was made repeatedly to coincide with some inborn reflex, it acquired the power of producing the same reaction as the unconditioned or inborn reflex. For example: food produced a certain reaction; the animal performed certain movements with respect to the food; saliva and gastric acids were secreted. This is an unconditioned reflex action, but if the feeding of the animal coincided several times with a certain sound, then this sound eventually evoked the same food reaction as the food itself, and became a conditioned reflex, the amount of saliva secreted being an indication of the strength of the intensity of the reaction. Similar principles applied to the formation of numerous conditioned reflexes from the external world allowed an organism to make adjustments by which it existed.

If, after the conditioned reflex was firmly established, the stimulus (a sound) was given but food was withheld, the sound, on repeated trials, gradually lost its effect until no response was obtained. This suppression of the reflex disappeared spontaneously after a variable period of time, extending from minutes in some cases to hours in others. This temporary suppression of the reflex was spoken of as inhibition. Inhibition may be internal or external, local or general, and may exhibit such special characteristics as delay and differentiation. It then becomes an extremely complicated process, such as one uses daily when one learns to inhibit or suppress extraneous noises which inevitably reach the sensorium; while one listens to the heart sounds or when one gazes into a microscope with both eyes open, one unconsciously suppresses all auditory and visual stimuli except those in which he is directly interested. It also accounts for the seeming higher intelligence and reasoning powers of trained circus animals whose performances are purely reflex phenomena obeying rigid physiologic principles.

From the beginning of his researches on conditioned reflexes, Pavlov was so troubled by the fact that his animals went to sleep during experiments that he began to make use of this somnolent condition to study the physiologic basis of sleep. As a result of many years of work he arrived at the hypothesis that inhibition, already referred to, ordinary sleep and hypnosis are one and the same process. Every conditioned stimulus, as soon as it is used alone and not accompanied by an unconditioned one, sooner or later leads to a drowsy, sleepy and lethargic state. Some conditioned stimuli produce sleep more quickly than others, and in a similar way some animals develop this property

more easily than others. As a rule, the rapidity with which sleep is induced is directly proportional to the length of time during which the conditioned stimulus acts alone unaccompanied by the unconditioned one. For instance, if a dog in which a conditioned reflex had been established was starved for twenty-four hours and the conditioned stimulus was allowed to act alone, the food reaction, both motor and sensory, was produced in about fifteen seconds while the dog remained wide awake and active. If the interval between the application of the conditioned stimulus and the giving of food (the unconditioned reflex) was lengthened to thirty seconds, the dog invariably became drowsy and eventually fell soundly asleep and had to be forcibly awakened to take food.

This fact may be explained in the following way: the isolated, concentrated stimulation of a certain group of cells of the cortex leads to their exhaustion and sleep, unless accompanied by stimulation of other cells. Normal sleep comes as an expression of the general exhaustion of all the cells of the cerebral hemispheres (Pavlov). The manner in which the inactive state spreads from the exhausted cells to involve the whole cerebral cortex is not clear. It may be assumed that the exhaustion of given cells leads in some unknown way to the formation of some substance or process which produces an inactive state in the already exhausted cells, thence spreads to involve other cells which have not yet been fatigued and ultimately produces sleep.

This same tendency to go to sleep was seen in other experiments in which conditioned stimuli were not used. Whenever a new indifferent stimulus was given to an animal, the animal responded by placing its sensory receptors in the best possible position for receiving such a stimulus; i. e., it looked toward a light, or cocked its ears in the direction of a sound. This is called the orientation reflex. If such a stimulus was repeated at frequent intervals the reflex gradually diminished until it disappeared, and unless different stimuli were given the animal became somnolent and fell asleep. If this experiment was repeated several times, the same stimulus being used, the animal could be made to go to sleep with absolute certainty. The sleep-inducing action of the conditioned stimulus and monotonous repetition of an indifferent stimulus is paralleled by the fact that uniform and monotonous surroundings usually predispose to sleep in persons who are not necessarily tired and may be the logic behind such time-honored methods of inducing sleep in cases of insomnia as counting imaginary sheep, watching clouds go by or listening to the imaginary patter of rain. This state, which in the case of conditioned reflexes leads to sleep, is exactly the same as that which leads to the development of inhibition. Further, whenever the inhibition is developed in a definite phase, sleep usually

follows unless special steps are taken to prevent it. Finally, cases occur in which either inhibition or sleep may develop, or in which inhibition may pass into sleep or sleep pass into inhibition. These are the facts that led Pavlov to conclude that internal inhibition and sleep are identical processes. An illustrative example will show this close relationship between inhibition and sleep.

If a dog is given food regularly thirty seconds after the application of the conditioned stimulus, saliva begins to flow from five to ten seconds after the stimulus is given. If this experiment is repeated day after day for weeks and months, the latent period of the conditioned reflex gradually increases to fifteen, twenty or twenty-five seconds until it reaches within a second or two of thirty seconds, when no secretion occurs and the dog becomes motionless and falls asleep. Here, internal inhibition has passed directly into sleep. The reverse of this is also true and sleep has been shown to pass into inhibition.

The basis of this hypothesis is that any more or less prolonged stimulus to the cortex, unless it is accompanied by stimuli to other parts, will ultimately and inevitably lead to somnolence and sleep. The conditioned reflex, even when it concerns such an important stimulus as food, will lead to sleep if it is allowed to play on an isolated area of the cortex without other accompanying stimuli. From the examples given and a thorough study of Pavlov's work, I am convinced of the identical nature of the two phenomena.

However, if sleep and internal inhibition are one and the same process, how does one explain the fact that inhibition is a constant and essential element of the active waking state, which serves the most delicate physiologic mechanism of adjusting the higher organism to its environment, while sleep is essentially an expression of inactivity and rest for the cerebral cortex? This seeming incompatibility may be overcome if one regards internal inhibition during the alert state as partial or scattered sleep, affecting only certain cellular groups, cutting out all stimuli except those that affect a certain area of the cortex, whereas sleep is a generalized and diffuse internal inhibition which has spread to involve the whole cerebral cortex as well as the lower centers of the brain. Thus internal inhibition during the alert state represents a regional distribution of sleep which is kept within bounds by the antagonistic nervous process of excitation.

From this point of view one sees that if one wishes to prevent inhibition from passing into sleep, one must use new and different stimuli, thereby producing fresh points of excitation on the cerebral cortex, which inhibits the spread of the internal inhibition already started. In the experimental work, when sleep was being established, a fresh external stimulus would immediately dispel it. This is seen

in every-day life; whenever one becomes sleepy, he gets up and moves around, washes his face in cold water, starts some new work or pinches himself, all of which merely establish new points of excitation in the cortex, thus preventing the spread of inhibition and development of sleep.

In this conception, one sees a brilliant example of the principle of economy of nature, for, if internal inhibition and sleep are the same, the mechanism that renders possible the highest and most complicated adjustments of which the organism is capable is so adjusted that while certain structures of the most essential and highly differentiated elements of the body, the nerve cells of the cerebral cortex, are undergoing activity, the remaining nerve cells not directly concerned are thrown into a state of inactivity and rest, allowing recuperation to take place.

In a previous discussion I tried to point out the close relationship between the cataplectic and sleeping attacks, the gradations that may occur between these two extremes and the transitional attacks in which cataplexy may pass into a sleeping state or in which the resistance of a sleeping attack will result in a cataplectic one. Since sleep appears as a result of internal inhibition, it does not seem at all illogical that the transitional states described may be due to the same thing, and if this is true, cataplectic attacks may also be the result of some form of inhibition. As far as I have been able to determine, Pavlov did not produce a typical cataplectic attack in any of his dogs, but he did produce a condition resembling catalepsy, which, as has been seen, closely resembles cataplexy.

In the experiments, two types of reaction were usually observed, the secretory reaction (flow of saliva) and the motor reaction (the dog seizing the food when it was offered). In the waking state both reflexes were active and effective, but as the dog passed into the sleeping state varying degrees of intensity of sleep were observed. First, the secretory reflex was lost, while the motor response remained, then the secretory response returned and the motor one was lost, and finally both reactions completely disappeared as the animal passed into deep sleep. On waking, the reflexes returned in the same manner but in the reverse order in which they had occurred formerly. Is there any similar reaction in the narcoleptic patients in whom sleep shows varying degrees of intensity? I think so. Some of the patients noticed that just as they were passing into the sleep state they suddenly became weak and powerless to move a muscle (abolition of motor response) until some external stimulus (a touch) was applied. Here it looks as though the inhibition of the sensory stimuli which occurs preparatory to sleep had spread to involve the motor area as well.

As has been seen, the production of sleep is favored by the lack of extraneous stimuli reaching the cortex, as well as by stimuli of a monotonous character. Such stimuli playing on an isolated area of the cortex result in fatigue, or sleep, and internal inhibition unchecked by fresh stimuli spreads to produce general sleep. In many of the narcoleptic patients there is such a mechanism at work, as in cases in which the monotonous stimulation of driving a car produced sleep unless the person stopped the car, got out and ran up and down the road a few times, thereby producing new points of excitation in the cortex which checked the spread of inhibition, thus preventing sleep. Other patients could prevent sleep by mental effort, physical exercise or by tensing their muscles. This also accounts for the ease with which these patients can be awakened, a slight touch, or stepping off the sidewalk if walking, being sufficient stimulation to overcome the inhibition present.

From a thorough study of Pavlov's discoveries, many of the characteristics of narcolepsy are elucidated. The occurrence of sleep as a result of few or monotonous stimuli reaching the cortex, the transitions from sleep to cataplexy, the varying degrees of the intensity of sleep, the ease with which sleep may be dispelled by the application of fresh stimuli, have all been duplicated in Pavlov's dogs, and it seems evident that narcolepsy is due to inhibition. The mechanism by which this inhibition is brought about may vary in different cases and may be either functional or organic.

SUMMARY AND CONCLUSIONS

The essential features of narcolepsy, as they have been developed during the course of this paper, may be reviewed and summarized as follows:

1. Narcolepsy is a condition characterized by the occurrence of sudden attacks of sleep and attacks of cataplexy.

2. While the attacks of sleep present many characteristics that are foreign to normal sleep, there is sufficient similarity between them to justify the clinical application of the term sleeping attack and the assumption that they may have a common etiologic mechanism.

3. All gradations between sleeping attacks without cataplexy to cataplexy without sleeping attacks may occur.

4. Narcolepsy occurs in both sexes and is essentially a chronic condition; although it may incapacitate the person so afflicted, it apparently does not shorten his life.

5. The sudden onset, brevity and rapidity of recovery of the two types of attack are sufficiently similar to hypothecate a common etiology.

6. The occurrence of dreams during the diurnal attacks of sleep, together with the marked nocturnal restlessness and vivid dreaming, indicates that one is dealing with a mechanism that is disturbed nocturnally as well as diurnally.

7. Narcolepsy may occur as a sequel of epidemic encephalitis.

8. The association of obesity with narcolepsy points to a disturbance of the vegetative centers of the brain and of the endocrine system.

9. Narcolepsy shows many clinical manifestations similar to other conditions, especially myoplegia and epilepsy.

10. Pavlov's work on conditioned reflexes with the production of sleep as a result of internal inhibition is reviewed, and the theoretical relation of this work to narcolepsy is discussed.

11. The relation to narcolepsy of the sleep that may be produced by organic lesions of the floor of the third ventricle and the aqueduct of Sylvius is discussed.

12. Attention is drawn to the marked loss of muscular tone and abolition of deep reflexes with the occurrence of an extensor plantar response in both cataplexy and normal sleep.

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THE USE OF SULPHUR FOR THE PRODUCTION OF FEVER*

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The element sulphur has had a long medical history, from its ancient rôle as a "spring tonic" to its modern scientific employment in baths and ointments for dermatologic conditions. Pharmacologists have denied that, taken internally, it has any other than a mild laxative action, which they attribute to the formation of small quantities of the irritating hydrogen sulphide. Sulphur applied to the skin is said to owe its effectiveness to the gradual production of hydrogen sulphide, which is poisonous to the infecting agent.

The injection of sulphur has a shorter history. As early as 1907, preparations of sulphur were made for subcutaneous injection by the French, chiefly Louis Bory,[1] Delehaye and Piot,[2] and Fleig.[3] Strangely enough, in view of the recent use of injections of sulphur to produce fever, these authors claimed that it reduced the temperature in cases of bronchopneumonia and pulmonary tuberculosis.

In 1921, Meyer-Bisch and Basch[4] reported the use of sulphur in injections of oil for the treatment for arthritis, and studied the abnormal metabolism occasioned by such injections. In 1927, Knud Schroeder[5] first used sulphur for treatment in dementia paralytica, although this had been suggested in 1925 by D. F. Tutunji.[6] Schroeder had turned to this pyrogenic agent in an effort to avoid the difficulties associated with malaria therapy. His technic called for the injection of from 1 to 2 cc. of a solution of 1 per cent sublimated sulphur in olive oil into the lateral

* Submitted for publication, Nov. 26, 1930.

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* Read at a meeting of the Chicago Neurological Society, Nov. 20, 1930.

1. Bory, Louis: Sur l'introduction du soufre dans l'organisme par la voie sous-cutanée, Compt. rend. Soc. de biol. **63**:512, 1907.

2. Delehaye and Piot: À propos de l'introduction du soufre sous la peau, Compt. rend. Soc. de biol. **63**:601, 1907.

3. Fleig, C.: Le soufre en nature, insoluble, colloidal ou à l'état naissant en injections sous-cutanées et intra-veineuses, Compt. rend. Soc. de biol. **63**:625, 1907.

4. Meyer-Bisch, R., and Basch, E.: Ueber das Schicksal parenteral verabreichten Schwefels und seinen Einfluss auf den Stoffwechsel, Biochem. Ztschr. **118**:39, 1921.

5. Schroeder, Knud: Ueber Schwefelbehandlung der Dementia paralytica und anderer metaluetischer Affektionen des Zentralnervensystems, Klin. Wchnschr. **6**:2189, 1927.

6. Tutunji, Djamil F.: Subcutaneous Injections of Colloidal Sulphur in Neuritis (Letter to Editor), Lancet **2**:408, 1925.

aspect of the thigh. Succeeding doses were given at intervals of four or five days, each dose being increased by 1 cc. until ten or more injections were given. The right and left thighs were used alternately, and the sulphur was injected underneath the fascia lata. W. H. Shilvock [7] reported good results from the use of an initial dose of 0.5 cc. of a 1 per cent solution of sublimated sulphur and an increase of only 0.5 cc. with each injection. In my experience, however, much better reactions were obtained when a 2 per cent solution of sublimated sulphur in olive oil was used, and each dose was increased by 1 cc. The U. S. Dispensatory [8] stated that sulphur is soluble in olive oil in the ratio of 1:100, but as a rule much of the sulphur in the 1 per cent mixtures remains undissolved at ordinary temperatures. With heating to about 150 or 180 F., which is advisable to facilitate the injection, most of the sulphur goes into solution, only to be reprecipitated as the solution cools while being placed in the syringe. It is never possible to dissolve all of the 2 per cent mixtures by heating.

CLINICAL OBSERVATIONS

Each of a series of injections of the 2 per cent solution of sublimated sulphur is followed by many physiologic changes that may be observed clinically. They may be described as follows:

1. *Temperature.*—Within from six to eighteen hours (usually twelve hours) after the injection, there is a sharp rise in the temperature, as measured by rectum, to a peak of from 103 to 105 F., followed by a fall to from 101 to 102 F. within three or four hours. Secondary elevations of temperature then characteristically occur daily or oftener for one or two days, so that the patient has a significant elevation of temperature for from thirty-six to forty-eight or even sixty hours. This febrile period is longer, therefore, than that brought about by any of the other methods of inducing fever, and herein lies the great therapeutic advantage of the method (fig. 1).

There is, in general, a relationship between the size of the dose and the height of the fever reaction, since larger doses produce higher temperatures. It is necessary, however, to increase the dose with each injection to obtain successively equal fever reactions, and even with a steadily and regularly increasing dose considerable variation occurs from one reaction to another. Also, different patients are unequally susceptible, some exhibiting a degree of refractoriness comparable to that seen in certain patients to injections of typhoid vaccine. In general,

7. Shilvock, W. H.: The Pyrogenetic Action of Sulphur in Olive Oil, Lancet **2**:347, 1930.

8. U. S. Dispensatory: Sulphur Sublimatum (Uses), ed. 21, Philadelphia, J. B. Lippincott Company, 1926, p. 1054.

those patients in whom typhoid vaccines easily produce fever reactions give good responses to the injection of sulphur. These variations from patient to patient and from time to time in the same patient are difficult to explain, but probably depend, at least in part, on individual idiosyncrasy and variations in the intake of water and in the insulation of the patient. Because of these facts, careful clinical observation and control are necessary for the proper determination of dosage.

2. *Chill.*—In spite of the rapid rise of temperature at the onset of the fever reaction, there is rarely a chill. The average patient fails to experience even a chilly sensation, but a few have had moderate or even severe rigors. The absence of the chill from the reaction in the vast majority of cases marks a distinct gain so far as the comfort of the patient is concerned.

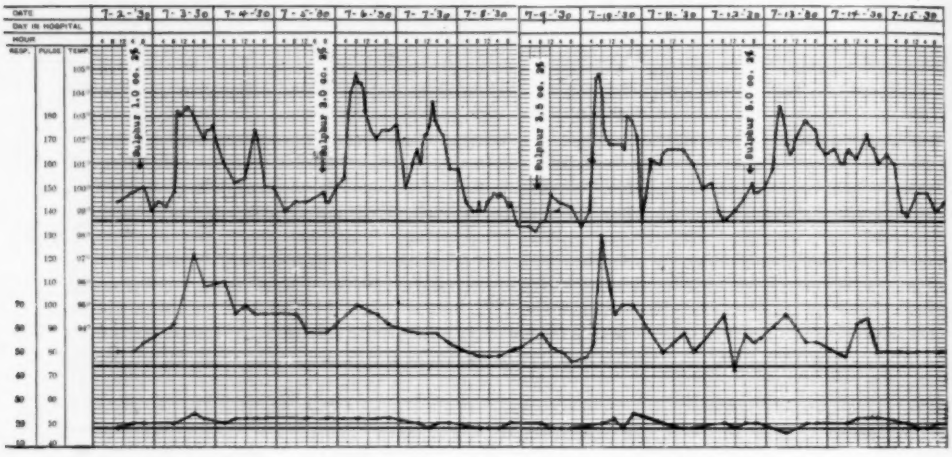

Fig. 1.—Clinical chart of temperature, pulse and respiration in a patient with dementia paralytica treated with injections of a 2 per cent solution of sublimated sulphur in olive oil. Note the variability of the pulse rate.

3. *Pulse Rate.*—Coincident with the increase of temperature, there is usually a definite rise in the pulse rate, but here again great variation is the rule. Some patients show a much higher pulse rate than one would ordinarily expect from the amount of fever present. For instance, one patient with a temperature of only 102 F. had a pulse rate of 132 (fig. 2). Other patients show little or no rise of pulse rate, as exhibited in a patient who, with a rectal temperature of 104.8 F., had a pulse rate of 96. Another patient with a temperature of 102.6 F. had a pulse rate of 80. The extent to which a patient shows elevation of the pulse rate is usually fairly constant in that patient from time to time. The pulse, in addition to being more or less elevated in rate, is variable

from one reading to another. For instance, one patient whose temperature at 4 p. m. on Aug. 19, 1930, was 99.2 F. by rectum showed a pulse rate of 104. At 8 a. m. on the next morning, the temperature was again 99.2 F. rectally, but the pulse rate was only 78. This quality seems inherent in the pulse curves in all patients treated with sulphur and seems to bespeak a highly irritable pulse-regulating mechanism. In all these changes in the pulse rate, the rhythm of the heart seems unaffected.

4. *Respiratory Rate.*—The respiratory rate is affected very little by the injection of sulphur, presenting no more variation than is commonly seen in the average patient in the hospital.

5. *Flushing and Sweating.*—The vasomotor and diaphoretic responses seem less marked than in patients receiving typhoid vaccine intravenously. While flushing and sweating occur, they are negligible. As

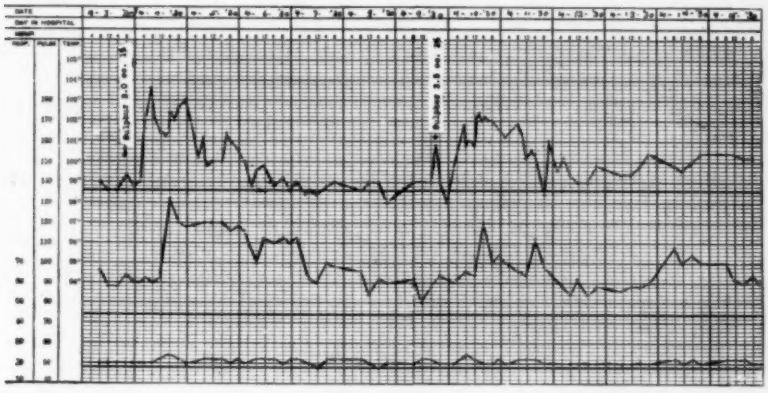

Fig. 2.—Clinical chart of temperature, pulse and respiration in a patient suffering from advanced multiple sclerosis treated with intramuscular injections of a 2 per cent solution of sublimated sulphur in olive oil.

a rule both the systolic and the diastolic blood pressure fall slightly, but this is not true in every case. For instance, in one patient the blood pressure was 106 systolic and 62 diastolic before injection, and during the height of the fever reaction it measured 120 systolic and 70 diastolic. No studies have been made as yet on patients with hypertension, nor have careful observations on temperature changes of the skin been recorded.

6. *Blood.*—Hematologic changes are of interest. There is a prompt leukocytosis, usually to from 20,000 to 30,000, with a polymorphonuclear percentage of from 80 to 90. Over a long series of injections, the red cell count seems to fall slightly, but the amount of decline is insignificant.

7. *Weight and Metabolism.*—There is usually a loss of from 10 to 15 pounds (4.5 to 6.8 Kg.) during a course of ten injections. Water is

apparently stored during a fever reaction, and excreted more rapidly during the period of decline of the temperature curve. Meyer-Bisch and Basch,[4] in 1921, carried on rather extensive investigations into the changes in the metabolism of nitrogen and sulphur, and found both to be excreted in increased amounts during and following fever. They also reported increased glycuronic acid and increased urobilin in the urine. These changes are for the most part only those that occur in any fever, and give but little clue to the manner in which sulphur brings about a febrile reaction.

8. *Local Pain.*—The comfort of the patient is but little affected by the fever. There is, however, pain at the site of injection for two or three days, chiefly on active movement of the limb. Local tenderness of moderate degree is the general rule. If the injection is made superficially, so that the sulphur is not placed beneath the fascia lata, marked heat, redness and local swelling occur. The pain many be controlled by codeine, but acetylsalicylic acid and other antipyretic drugs are best avoided, since they reduce the temperature and defeat the purpose of the treatment.

The matter of local pain on movement assumes considerable importance in some patients, especially in those with an unclouded mentality and a tendency to depression. A few such patients have made serious objection to it. However, the usual patient with dementia paralytica seems but little troubled, because of the euphoria or mental obfuscation so often present. In patients other than those with dementia paralytica, the pain may render the use of sulphur inadvisable.

MECHANISM OF FEVER PRODUCTION

Two questions present themselves to the physician using sulphur for the production of fever. The first relates to the mechanism by which fever is produced, and the second concerns the reason for the clinical improvement in the patient. It is strange that the recent literature on the use of sulphur as a pyrogenic agent, though somewhat voluminous, especially in German, contains no report of any experimental work. Many clinical reports are available,[9] describing the pyrexia and discussing the therapeutic effects obtained, but there is no attempt to explain why fever occurs, and nothing but speculation as to the

9. Marcuse, Harry, and Kallmann, Franz: Zur Sulfosinbehandlung der Paralyse und Schizophrenie, Nervenarzt **2**:149, 1929. Harris, Noel G.: Treatment of General Paralysis of the Insane by Injection of Sulphur, Lancet **1**:1068, 1930. Patterson, William G., and Switzer, S. R. L.: Sulfosin Therapy in General Paralysis of the Insane, Lancet **2**:348, 1930. Winkler, L.: Ueber Schwefeltherapie der Optikusatrophie, Wien. klin. Wchnschr. **41**:374, 1928. Read, Charles F.: A Fever Treatment of Dementia Paralytica by Injection of Sulphur in Olive Oil, Arch. Neurol. & Psychiat. **24**:649 (Sept.) 1930.

cause of the improvement in the condition of the patient. According to Schroeder,[5] H. M. Meyer held that the general effect of injection of sulphur is bound up with its "colloid" nature, and depends not on sulphur itself but on "the intramuscular injection of sulphur." Meyer-Bisch [10] thought that a fundamental change occurs in the metabolism of endogenous albumin. Spiro [11] suggested a possible formation of a sulphur-albumin compound. Schroeder himself held that sulphur, being an "avid chemical," has a specific or chemotherapeutic effect on late syphilitic lesions, apart from its fever-producing effect.[12] So far as I know, none of these theories is based on experimental evidence. The present article is a report of work done in an effort to elucidate this matter as to how sulphur produces its effect. The experiments are not yet complete, but are advanced sufficiently to warrant their description and consideration.

Six rabbits in apparent good health were selected, each weighing as nearly 5 pounds (2.3 Kg.) as possible. For a week the rectal temperature of each rabbit was taken twice daily. The result was surprising in that the rabbits' normal rectal temperature was found to vary unpredictably between 102 and 105 F. This variability could not be correlated with any environmental factor or any change in the rabbits' physiologic state.

With this, then, as a standard of normality for the rabbits' rectal temperature, 0.25 cc. of a 1 per cent solution of sublimated sulphur in olive oil [13] was injected intramuscularly into one of the rabbits at 6 p. m., and the temperature was taken every two hours the next day. This rabbit received more than 7.5 times as much sulphur per pound of body weight as is given a man for the first dose, and yet no elevation of the rabbit's temperature was detectable twelve hours after the injection, or for the next twelve hours. The next rabbit was consequently given 1 cc. of the 1 per cent solution of sublimated sulphur in olive oil, which is the initial dose for a man weighing 30 times as much. Still no detectable fever occurred. Neither did the rabbit give evidence of any great discomfort.[14]

Biopsies were then made on several injected rabbits at varying intervals after the injection. For at least a week following the injection, the rabbits' muscles were felt to contain a firm mass, and when a muscle was exposed at operation it was seen to be swollen and irregularly hyperemic and congested, rather than semitranslucent as is normally the case. Gross sections of these muscles showed hyperemia and in addition a yellow mottling which extended

10. Meyer-Bisch, Robert: Ueber die Behandlung chronisch deformierender Gelenkerkrankungen mit Schwefel, Klin. Wchnschr. **1**:575, 1922.

11. Spiro, Paul: Zur unspezifischen Reizkörpertherapie chronischer Gelenkerkrankungen, Deutsche med. Wchnschr. **53**:914, 1927.

12. Schroeder, Knud: Ueber Sulfonsinbehandlung von Nervensyphilis und anderen syphilitischen Erkrankungen, Klin. Wchnschr. **7**:1636, 1928.

13. The preparations of sulphur in olive oil used in this study were supplied by Abbott Laboratories, Chicago.

14. In an effort to produce fever in rabbits by other methods, the intravenous injection of neither 1,000,000 typhoid-paratyphoid bacilli nor of 0.5 cc. of boiled milk was successful. The temperature-regulating mechanism in the rabbit is evidently very poorly organized.

irregularly throughout the muscles and especially along the septums of the muscles.

Microscopic sections, made twenty-four and forty-eight hours after injection of sulphur, when stained with hematoxylin-eosin and with phosphotungstic acid, showed a profound necrosis and an inflammatory reaction. As viewed under a low power magnification, those areas which in the gross appeared yellowish were seen to take the phosphotungstic acid stain very poorly. These poorly stained regions were closely packed with polymorphonuclear leukocytes, a few mononuclear cells and much amorphous débris (fig. 3). Under a somewhat higher magnification, engorgement of blood vessels and hemorrhages were seen. The

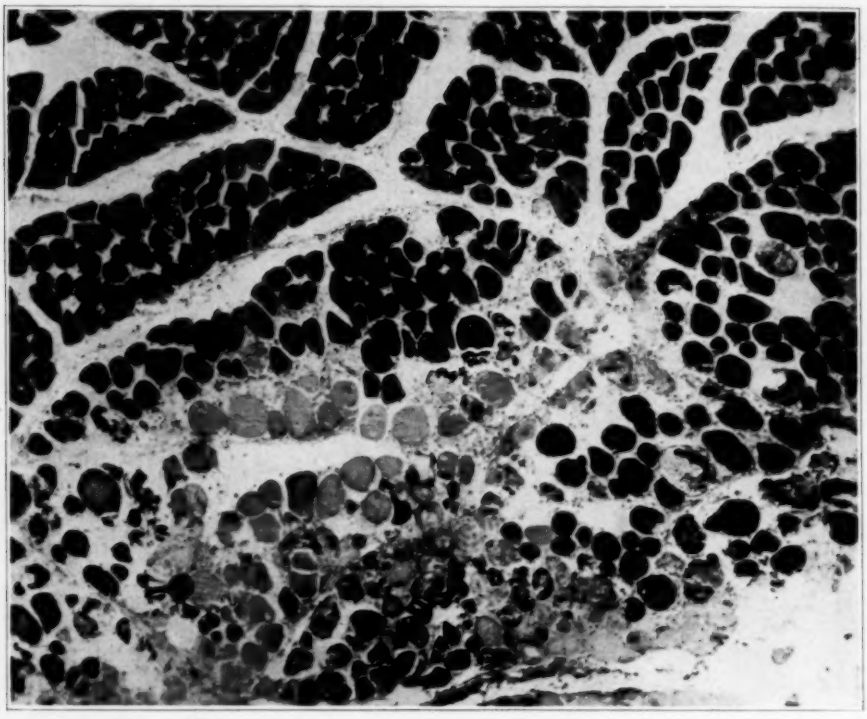

Fig. 3.—Photomicrograph of a cross-section of rabbit muscle twenty-four hours after injection of 0.5 cc. of a 1 per cent solution of sublimated sulphur in olive oil. Note the poor staining and fragmentation of necrotic fibers, infiltration with inflammatory cells and collections of débris. Phosphotungstic acid; × 60.

muscle bundles in these necrotic areas showed a marked loss of normal striation, and in places, extreme fragmentation and degeneration of the muscle tissue were observed. In the midst of areas showing the most profound necrosis, many muscle fibers had been invaded by great numbers of inflammatory cells, leaving only the vague outline of the original muscle structures (fig. 4). In all these sections I sought in vain for evidences of sulphur.

Thirteen days following injection, most of the acute inflammatory process had subsided and a marked proliferation of fibroblasts had occurred. Numerous

regenerating muscle fibers and many so-called foreign body giant cells were also seen, while most of the tissue débris seen in earlier sections had disappeared (fig. 5). These gross and microscopic evidences of inflammation in the muscles of the rabbits are strikingly consistent with the heat, redness, swelling and local pain and tenderness noted clinically when the injection is made too near the surface.

Control experiments were designed to determine whether the olive oil was essential to the reaction. A 1 per cent suspension of sulphur was made in a mixture of 1 per cent acacia in physiologic solution of sodium chloride. The acacia was necessary to hold the sulphur in a fine state of suspension. When this

Fig. 4.—Photomicrograph of longitudinal section of rabbit muscle forty-eight hours after injection of 0.5 cc. of a 1 per cent solution of sublimated sulphur in olive oil. Note the extreme necrosis, with loss of structure of fibers, massive polymorphonuclear invasion and interstitial hemorrhage. Phosphotungstic acid; × 230.

preparation, which was sterilized in a water bath or in the autoclave, was injected into the human subject, fever and the other reactions were noted just as after injections of sulphur in olive oil. When this suspension of sulphur and acacia was injected into a rabbit, the same pathologic picture was found as after injections of sulphur in olive oil (fig. 6). These experiments with sulphur in acacia demonstrate that the olive oil is not essential to the fever reaction. On the other hand, Shilvock has stated that sulphur in water failed to produce fever.[7] Sulphur in a preparation of acacia and saline solution differs from sulphur in water only,

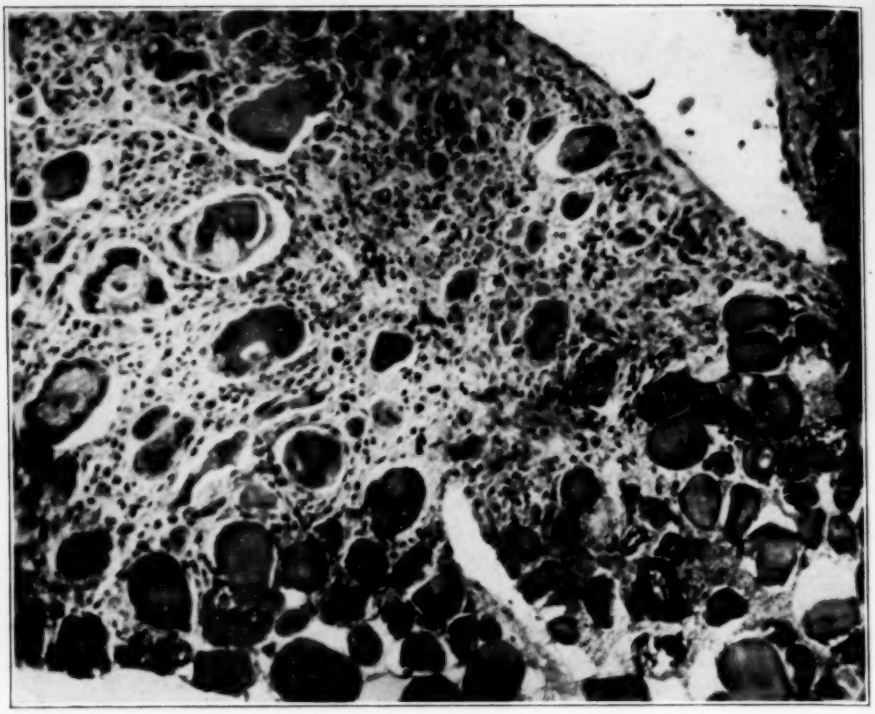

Fig. 5.—Photomicrograph of a cross-section of rabbit muscle thirteen days after injection of a 1 per cent solution of sublimated sulphur in olive oil, showing "foreign body giant cells," proliferation of fibroblasts and regeneration of muscle fibers. Hematoxylin-eosin; × 145.

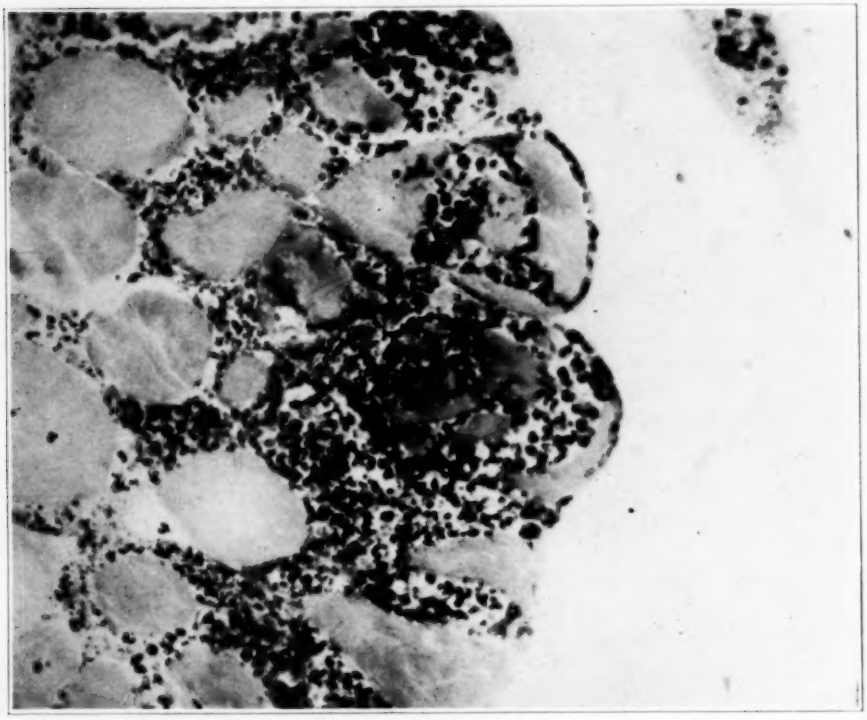

Fig. 6.—Photomicrograph of a cross-section of rabbit muscle forty-eight hours after the injection of physiologic solution of sodium chloride containing 1 per cent each of acacia and sublimated sulphur. Note necrosis of muscle fibers with infiltration by polymorphonuclear leukocytes and accumulation of débris. Hematoxylin-eosin; × 325.

in that the acacia holds the sulphur in a fine suspension. It seems, therefore, that it is the physical state of fine division and suspension of the sulphur that is favorable to the reaction, rather than that the olive oil itself is necessary, or that a chemical union necessarily occurs between the sulphur and the oil.

COMMENT AND THEORY

This work, conceived in an effort to demonstrate the manner in which injections of sulphur cause fever, has not progressed sufficiently to allow me to draw final conclusions. It seems likely, however, that the fever occurs as a result of a protein reaction, the protein being liberated from the patient's own muscle tissues by sulphur, or by the irritant hydrogen sulphide formed from sulphur in the tissues. The following four considerations render this explanation probable: 1. The fever does not occur until about twelve hours after the injection. This latency argues for some interaction between the sulphur and the tissues before fever can occur. 2. It should be noted that increasing doses of sulphur are required to produce successively equal fever reactions. This necessity for increasing the dose is the one most striking characteristic of the fever produced by injections of protein. If the fever were a "chemical fever," or if sulphur acted pharmacologically rather than biologically, one would expect equal doses to have equal effects. The building up of an immunity to sulphur strongly suggests that the reaction is a biologic one. 3. As already mentioned, patients in whom it is easy to produce fever by the injection of typhoid vaccines also give good fever reactions to injections of sulphur. According to my observation, the opposite is true also, that patients respond poorly to sulphur who respond poorly to typhoid vaccine. This is a rather definite similarity that seems to speak for a resemblance in the physiologic mechanisms involved. 4. Most conclusive of all, the injection is attended by the formation of a necrotic area with the destruction of muscle tissue and a marked inflammatory reaction. It is difficult to see how such a destruction of tissue can fail to produce fever. These facts, while perhaps not conclusive or final, seem to indicate that the physiologic response to injections of sulphur depends on the gradual absorption of proteins or protein fragments from the patient's own tissues. They place the fever following injections of sulphur on the same basis as that following extensive deep roentgen therapy or that following extensive trauma to living tissue.

Furthermore, these results, while not bearing directly on the question, would seem to throw doubt on the thesis held by Schroeder, that sulphur exerts a specific, chemotherapeutic effect on syphilis. They indicate rather that any beneficial effects that are produced in dementia paralytica and other syphilitic manifestations are attributable to the fever, in the same sense that one attributes improvement to the fever of malaria or injections of *Bacillus typhosus*; whether it is actually the

fever or rather the whole constitutional response to protein that proves beneficial, it is not in the province of this paper to answer.

The question of the therapeutic value of the treatment is to be left unanswered in this article. To answer it with any degree of accuracy and dependability will require much more time than has yet elapsed since this use of sulphur was made available to American physicians. I have had under my care several patients with dementia paralytica, some of whom made excellent progress while being treated in this way. Others treated have had multiple sclerosis, encephalomyelitis disseminata and the meningovascular type of syphilitic infection of the central nervous system.

At the present time, one can say that in the treatment for these conditions, the injection of sulphur can accomplish what fever produced by any other method can accomplish, and that by virtue of the long duration of the fever produced sulphur should bring results more rapidly and surely than any other method. It seems superior to typhoid-paratyphoid vaccine in that the fever is of longer duration and in that the chill is usually avoided, and inferior in that the local pain and tenderness may be troublesome in patients in whom the pain threshold is low, or who are already depressed.

The localized necrosis produced by the injection of sulphur seems to heal readily and leave no discernible defect in the function of the muscles. However, it will be understood from the histologic changes illustrated here that a series of ten injections of sulphur, particularly the final doses of 8, 9 or 10 cc., must destroy a great deal of muscle tissue. Especially would this be true should more than one series be given, with the result that after many injections sufficient fibrosis and scarring might be produced in the muscles to impair their function. Consequently, it is my opinion that the use of sulphur as a pyrogenic agent should not be undertaken lightly or in cases in which the therapeutic result promises to be slight. It would seem to be applicable chiefly in cases of dementia paralytica in which malaria therapy would otherwise be used. It has several advantages over malaria, however, in that it produces fever of longer duration; that it has, so far as is known, no mortality, and that it is easier to control clinically. In fact, it gives a safe and convenient method of producing therapeutic fever to the average physician who has too few patients with dementia paralytica to permit him to maintain a strain of the plasmodium, and who has inadequate facilities for handling patients with malaria. Because of these facts, and with the reservations specifically mentioned, it is believed that the use of sulphur administered intramuscularly is a safe, effective and convenient addition to our therapeutic armamentarium.

SUMMARY

1. Sulphur in olive oil, when injected into the muscles of the outer part of the thigh, produces a high fever, lasting from thirty-six to sixty hours, with variable elevation in the pulse rate, a vasomotor and a diaphoretic response less marked than in fever from typhoid vaccine, a marked polymorphonuclear leukocytosis and other less important physiologic changes. Local pain and tenderness at the site of injection, while present, are not particularly troublesome to the average patient with dementia paralytica.

2. Experimental injections of suspensions of sulphur in oil and sulphur in acacia-saline solution into the rabbit's leg show a marked localized necrosis with an acute inflammatory reaction which is finally resolved. This work tends to show that the fever and other changes following the injection of sulphur intramuscularly result from the breaking down of muscle tissues into fragments which, when absorbed, cause a so-called "protein reaction."

3. Fever produced by injections of sulphur seems capable of effecting the same therapeutic results that any other form of fever can produce. It is chiefly of value in cases of dementia paralytica in which malaria is indicated, and in many ways is superior to malaria, especially in the hands of the average physican.

8 South Michigan Avenue.

DISCUSSION

DR. S. HIRSCHFELD: What was the average number of injections per course?

DR. JOHN FAVILL: The value of this treatment will be settled when it is determined how much fever is necessary to bring about a therapeutic effect in these cases. If, for instance, a temperature over 102 F. is sufficient, then I think that the decline by lysis which occurs with this treatment is bad, is debilitating to the patient and wastes time. I hope that it may be brought out in the discussion just what level of temperature is of value.

DR. GEORGE W. HALL: Dr. Mackay forgot to mention that the temperature following injections of sulphur does not begin to rise immediately as in injections of typhoid vaccine.

I think that the remarks of Dr. Favill are timely, for the more I have to do with these cases, the more I am inclined to think that one must induce high temperatures to obtain results; it is a question whether a temperature of from 101 to 103 F. produces the results desired. The disadvantage, of course, is that the patient must be kept in the hospital for a long time to receive the proper number of injections. It takes almost twice as long to give the required number of injections of sulphur as it does in the use of typhoid and paratyphoid vaccines. It seems that the temperature due to injections of typhoid and paratyphoid can be somewhat better regulated. As Dr. Mackay said, patients do not object so much to the chills produced by typhoid vaccine as they do to the pain of the injections of sulphur. That is especially true in the treatment of cases of multiple sclerosis, while persons with dementia paralytica are more or less indifferent in their reaction.

DR. CLARENCE A. NEYMANN: The question of the height of the temperature to be of therapeutic value has been investigated by us, and we think that a temperature ranging between 103 and 105 F. is the most suitable.

A sure method of regulating the fever is by the use of diathermy. In this way one can produce any degree of fever desired for any period of time. In the cases in which the fever curve rose and fell abruptly, we had fair results; however, when we sustained the temperature for approximately nine hours and gave a series of treatments, our results improved. Thus we can almost predict improvement in every grandiose patient who is not demented.

DR. A. B. YUDELSON: A man under 30, with multiple sclerosis, was given injections of 1 per cent sulphur in oil at the County Hospital. Eight hours afterward a temperature of 102 F. developed, which was sustained for about eight hours. Hiccups then developed which became so violent that we feared death. Morphine was given to relieve the spasms, and the patient survived. He is still alive. I wondered at the time whether or not multiple sclerosis is a condition in which sulphur and oil is contraindicated.

DR. R. P. MACKAY: In this work ten injections usually constituted a course, because by that time a dose of 10 cc. had been reached, and we thought it unwise to use greater amounts. Many times a patient can be given a second series after several weeks, if it is desirable.

I believe that the latency before the onset of fever and the slow decline of temperature are probably due to the fact that muscle tissue is slowly broken down and absorbed. A certain amount of time is necessary for the sulphur (or perhaps hydrogen sulphide formed from sulphur) to break down muscle tissues. On the other hand, in cases in which intravenous injections of typhoid vaccine are used, the protein injected is already "dead" and perhaps already fragmented, so that it is in a position to produce immediate fever. The reaction consequently occurs within an hour or two.

As to whether time is wasted during the prolonged period of lysis when the temperature is falling to normal, it should be remembered that during most of this time the temperature is above 102 F. If temperatures at or above 102 F. are of value in the treatment of dementia paralytica, little time is lost during lysis, since the fall is rapid below that point. In the average reaction the temperature is above 102 F. during probably from twenty to thirty hours. This constitutes a longer period of therapeutic fever than that produced by any other method with which I am familiar. Even nine hours of fever produced by diathermy compares rather poorly with twenty or thirty produced by sulphur.

As to the prolonged period in the hospital which is necessary in order to produce ten reactions in the patient, it seems to me that if it is the fever that is benefiting the patient, the number of injections is not important. For instance, if ten injections of typhoid vaccine give a total of forty hours of fever above 102 F., and if an elevation of temperature for the same length of time can be obtained with one or two injections of sulphur, why should not one or two injections of sulphur be equivalent to ten of typhoid vaccine? Thus, during even a limited period of hospitalization, it should be possible to produce a longer period of pyrexia with sulphur than with typhoid vaccine.

We have had no cases of multiple sclerosis treated with sulphur in which hiccups or other involvement of the diaphragm developed. In the case mentioned in the discussion I would blame the multiple sclerosis for this symptom rather than the sulphur.

I will add that the blood of one of our patients was examined spectroscopically for sulphemoglobin, but the characteristic absorption band could not be identified.

ENCEPHALOGRAPHIC STUDIES IN CRYPTOGENIC EPILEPSY *

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With the advent of the encephalographic method a new avenue of approach was opened for the investigation of convulsive states. In 1923, Dandy [1] drew attention to the characteristic appearance of the dilated subarachnoid spaces which form pools of fluid overlying the gyri in some cases of epilepsy. He also noted the dilated ventricles in these cases with the ventriculographic method which he originated. Since then, Foerster [2] has reported dilated asymmetrical ventricles and accumulation of air over the cortex in eleven cases. On the other hand, Tyczka [3] found abnormal conditions in only two of eighteen cases studied by the encephalographic method. There are also reports by other workers, among them Schuster,[4] Fischer,[5] Carpenter,[6] Bingel.[7] Waggoner,[8] Wartenberg,[9] and Friedman, Snow and Kasanin.[10] Fay [11]

* Submitted for publication, Oct. 22, 1930.

* From the Manhattan State Hospital, New York.

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9. Wartenberg, R.: Encephalographische Erfahrungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:585, 1924-1925.

10. Friedman, E. D.; Snow, W., and Kasanin, J.: Experiences with Encephalography Via the Lumbar Route, Arch. Neurol. & Psychiat. **19**:762 (May) 1928.

11. Fay, Temple: Some Factors in the "Mechanical Theory of Epilepsy," with Especial Reference to the Influence of Fluid, and Its Control, in the Treatment of Certain Cases, Am. J. Psychiat. **8**:783, 1929.

recently reported abnormal conditions in fifty-nine cases of epilepsy by the same method. He stated: "There has been noted a characteristic picture obtained by roentgenograms in those patients showing convulsive state . . . a definite change can be demonstrated in the fluid pathways by this method." He believed that there is an increased accumulation of cerebrospinal fluid in cases of epilepsy, owing to faulty drainage caused by the pathologic process of the pacchionian bodies. On this basis he developed the "mechanical theory of epilepsy," and advocated treatment by the method of dehydration.

One of the most serious criticisms that can be advanced against the use of encephalography in epilepsy is the question: What is known of the encephalographic conditions of the normal brain? One cannot offer as controls encephalographies on normal healthy persons, but the criticism can perhaps be met by encephalograms made on persons in whom pathologic changes in the brain were suspected but not found. A number of such encephalograms have been reported in the literature; in them the ventricles are not dilated and are symmetrical in size and position; there are no air pockets in the frontal, temporal or parietal regions. A number of encephalographic studies have also been made in patients with schizophrenia, but I would not suggest their use for a comparative study, as the pathology in schizophrenia is still debatable. A dilated ventricle, however, and a large accumulation of air in the subarachnoid spaces in a roentgenogram must be accepted as pathologic. The present question is: How frequently does one find these abnormalities in patients suffering from convulsive states? Are they constant factors, as Fay and others claim? To answer these questions I have undertaken encephalographic studies in a number of patients with epilepsy at the Manhattan State Hospital. I have selected especially the so-called idiopathic or cryptogenic cases, without focal signs, in which none or only very discrete neurologic symptoms are present.

I have performed encephalography on seventeen women between the ages of 18 and 49 years. The majority were in an advanced state of mental deterioration; one was mildly deteriorated and three showed no evidence of deterioration. The latter were committed to the hospital because of transitory confusional or dazed states following repeated convulsions.

I cannot reproduce here all the encephalograms. I shall, however, briefly report the observations in all cases and give a few characteristic illustrations. I took roentgenograms twenty-four hours after encephalography in all cases. In some of the cases the disappearance of air was very slow, and I shall give, therefore, a few illustrations of roentgenograms taken twenty-four, one hundred and twenty and two hundred and sixteen hours later. None of the patients died as a consequence of

the encephalographic procedure. Two died several weeks later, one after repeated severe convulsions and the other after a sudden hemoptysis.

METHOD

On the day of the encephalography, the patients had the usual breakfast at 7 a. m., but no midday meal. One hour prior to the lumbar puncture, which was done at 2 p. m., ⅛ grain (0.008 Gm.) of morphine and 1/150 grain (0.0004 Gm.) of scopolamine hydrobromide were injected subcutaneously.

No special apparatus was used. The usual lumbar puncture needle and a 20 cc. Luer's syringe were employed. In order to prevent the escape of air, the end-piece of the syringe which was introduced into the lumbar puncture needle was coated with sterile petrolatum. The patients were placed in a sitting position, and puncture was made between the third and fourth or fourth and fifth lumbar vertebrae. At first the fluid was withdrawn in amounts of 5 cc., replacing it by an equal volume of air. The amount of fluid withdrawn at one time was increased gradually, but it never exceeded 10 cc. The total amount of fluid drained and air injected ranged between 75 and 155 cc. A volume of air was injected corresponding to the amount of fluid withdrawn, but I believe that it would be advisable to reduce the total amount of air injected 10 cc., as the air at body temperature expands and thus probably adds to the pressure in the brain and increases the meningeal signs to which reference will be made later. The amount of fluid withdrawn was practically all that could be drained.

In order to distribute the air in all ventricles the patient's head was bent several times in various directions shortly after the cessation of the drainage. The patients were then immediately taken to the x-ray room. The encephalograms were taken with the patient in the recumbent position; first the lateral, then the anteroposterior and postero-anterior views. In the anteroposterior position, the head rested on a triangle at about 35 degrees incline; in the postero-anterior position, the forehead, nose and chin rested on the table.

REPORT OF CASES AND OBSERVATIONS

The encephalographic observations may be classified in three groups: (1) patients presenting normal appearances; (2) those with pronounced dilatation of the ventricles and of the subarachnoid spaces and (3) those with appearances that point to some pathologic change different from that in group 2.

Group 1: Patients with Normal Encephalographic Appearances.—Case 1.—J. G., aged 39, had had convulsions since the age of 4 years. Neurologic examination revealed somewhat irregular pupils and a tremor of the fingers. Mentally, the patient was not deteriorated. Encephalography was done on Sept. 26, 1929, and 120 cc. of fluid was drained and replaced by an equal volume of air.

Encephalography showed a moderate accumulation of air in the frontal and temporal regions; the lateral ventricles in the lateral views were normal in size; in the anteroposterior and postero-anterior views the lateral ventricles were slightly asymmetrical in position. There was a slight accumulation of air in the pacchionian area. The cisternae were well filled; the falx was well outlined; the third and fourth ventricles were seen. Traces of air were still present after twenty-four hours. The impression was that the encephalographic appearances were normal.

Case 2.—C. Z., aged 40, had had convulsions since the age of 24 years. She had unequal pupils, and was mentally deteriorated. Encephalography was done on Aug. 8, 1929, and 95 cc. of fluid was replaced by an equal volume of air.

Encephalography showed an accumulation of air in the frontal and temporal regions; the lateral ventricles were of normal size in all views; there was a slight accumulation of air in the area of the pacchionian bodies. The sulci were well seen, the cisternae were well outlined, and the falx was seen; the third and fourth ventricles were vaguely outlined. Only traces of air were observed after twenty-four hours. The impression was that the encephalographic appearances were normal.

Case 3.—P. S., aged 30, had had convulsions since the age of 9 months. The pupils were unequal, and there was pronounced mental deterioration. Encephalography was done on June 13, 1929; 135 cc. of fluid was obtained and replaced by air.

Encephalography showed a moderate accumulation of air in the frontal, temporal and occipital regions; the lateral ventricles were normal in size in the lateral views, but slightly asymmetrical in position in the anteroposterior and postero-anterior views. There was a moderate accumulation of air in the area of the main pacchionian bodies, and the sulci were well outlined. The cisternae were filled; the third and fourth ventricles were seen. There was a considerable disappearance of air after twenty-four hours. The impression was that the encephalographic appearances were normal.

Case 4.—R. H., aged 40, had had convulsions since the age of 19 years. She had chronic arthritis, with articular deformities. Mentally, she was mildly deteriorated. Encephalography was done on July 11, 1929, and 135 cc. of fluid was drained and replaced by air.

Encephalography showed a slight increase in the amount of air in the frontal and temporal regions and a slight accumulation in the occipital region. The lateral ventricles were of normal size. The sulci were well outlined; there was a moderate accumulation of air in the pacchionian area. The cisternae, falx and the third and fourth ventricles were outlined. There was a considerable disappearance of air after twenty-four hours. The impression was that the encephalographic observations were normal.

Case 5.—E. B., aged 39, a colored woman, had had convulsions since the age of 26 years. There were signs of vasomotor instability, with periodic edema of the lower eyelids; mentally, she was deteriorated. Encephalography was done on May 29, 1929, and 130 cc. of fluid was drained and replaced by an equal volume of air.

Encephalography showed a moderate accumulation of air in the frontal, temporal and perhaps occipital regions; the ventricles were of normal size in the lateral (fig. 1*A*) and anteroposterior views (fig. 1*B*). There was a very small accumulation of air in the region of the main pacchionian bodies; the sulci were marked; the cisternae were seen, and the third and fourth ventricles were outlined. There was a considerable disappearance of air after twenty-four hours. The impression was that the encephalographic appearances were normal.

Case 6.—Y. G., aged 39, had had convulsions since the aged of 12. There were no neurologic signs and no evidence of deterioration. She had been committed to the hospital because of a confusional state after repeated convulsions. Encephalography was done on Aug. 29, 1929, and 125 cc. of fluid was withdrawn and replaced by air.

Encephalography showed a slight accumulation of air over the frontal, temporal and occipital regions. The lateral ventricles were of small size. There was a small accumulation of air in the region of the pacchionian bodies; the falx was not outlined; the cisternae were seen, and the third and fourth ventricles were normal (fig. 2*A*). In the postero-anterior view the lateral ventricles were slightly asymmetrical in position (fig. 2*B*). There was considerable disappearance of air after twenty-four hours.

CASE 7.—F. H., aged 27, had had convulsions since the aged of 16. There were no neurologic signs. She showed profound mental deterioration. Encephalography was done on July 25, 1929, and 120 cc. of fluid was withdrawn and replaced by air.

Encephalography showed a moderate accumulation of air in the frontal and temporal regions, and a slight accumulation in the area of the main pacchionian bodies; the lateral ventricles were of normal size and symmetrical as seen in the

Fig. 1 (case 5).—*A*, lateral view of a normal encephalogram. The lateral ventricle is of normal size. There was slight accumulation of air in the area of the main pacchionian bodies, and moderate accumulation of air in the frontal, temporal and perhaps occipital regions. The cisterna chiasmatis and cisterna pontis are seen. *B*, anteroposterior view of the same encephalogram. The lateral ventricles are symmetrical in size and position. The third and fourth ventricles are well outlined.

postero-anterior view. The sulci were not well outlined; the cisternae were seen; the falx was not outlined, and the third and fourth ventricles were seen vaguely. Air was still present after twenty-four hours. The impression was that the encephalographic appearances were normal.

CASE 8.—M. R., aged 36, had had convulsions since the age of 10. She had an uneventful infancy. The pupils were slightly unequal. Mentally, she was deteriorated. Encephalography was performed on Sept. 12, 1929, and 125 cc. of fluid was replaced by air.

Encephalography showed a slight accumulation of air in the subarachnoid spaces in the frontal and occipital regions; the sulci were well marked; the lateral ventricles were of normal size, and the cisternae were clearly seen. Considerable air was present after twenty-four hours.

Summary.—In eight patients, 47 per cent, the encephalographic appearances were normal. The amount of air injected ranged between 95 and 135 cc. None of the patients showed any definite neurologic signs; five were definitely deteriorated, one only to a mild degree. The ages ranged between 27 and 40, but most patients in this group were in the fourth decade. With a few exceptions, there was considerable disappearance of air after twenty-four hours.

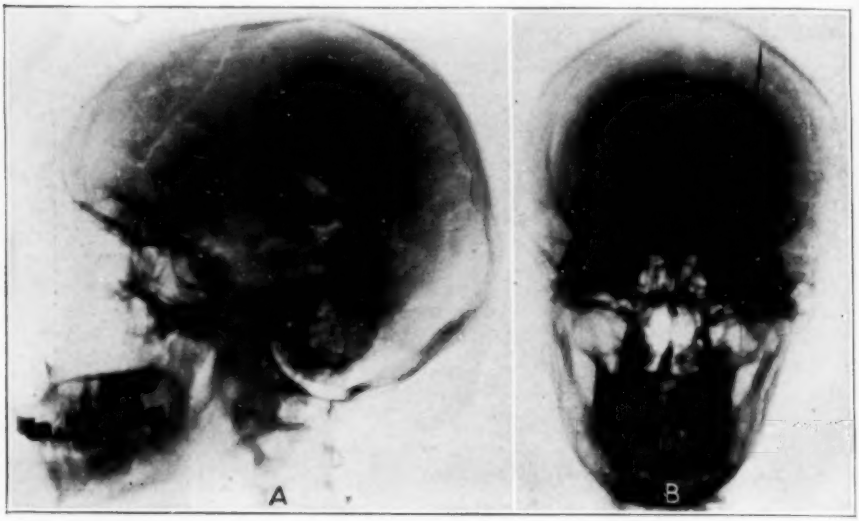

Fig. 2 (case 6).—*A*, lateral view of a normal encephalogram. Note the small size of the lateral ventricles, and the moderate accumulation of air in the frontal and temporal regions. The cisterna chiasmatis is well seen. There are fine air markings over the parietal region. *B*, postero-anterior view. A very slight asymmetry, but normal size of the lateral ventricles is seen.

If there is any question as to the interpretation of the observations in group 1, there will be none as to the dilatation of the ventricles and the accumulation of air in the subarachnoid spaces, giving the impression of internal and external hydrocephalus, in the following group of four cases.

Group 2: Patients with Dilated Ventricles and Large Accumulations of Air in the Subarachnoid Spaces.—Case 9.—A. B., aged 30, had had convulsions since the age of 12. She had had no serious illnesses. She had dilated pupils, a marked tremor of the fingers and general hyperreflexia. Mentally, she was confused and

had frequent hallucinatory episodes of excitement. Encephalography was performed on May 23, 1929, and 125 cc. of fluid was withdrawn and the same volume of air injected.

Encephalography showed a relatively moderate accumulation of air in the frontal and temporal regions and in the area of the main pacchionian bodies. The lateral ventricles, both in the lateral and anteroposterior views (figs. 3*A* and *B*) were decidedly dilated. All the horns of the lateral ventricles were well seen. The sulci, however, were not as marked as I have seen in other cases. I have the impression that there is a relatively lesser accumulation of air in the subarachnoid spaces in comparison with the increased amount of air in the ventricles. There seem to be leptomeningeal adhesions, with occlusion of the subarachnoid spaces, in some places. After twenty-four hours, roentgenograms showed that the same amount of air was still present.

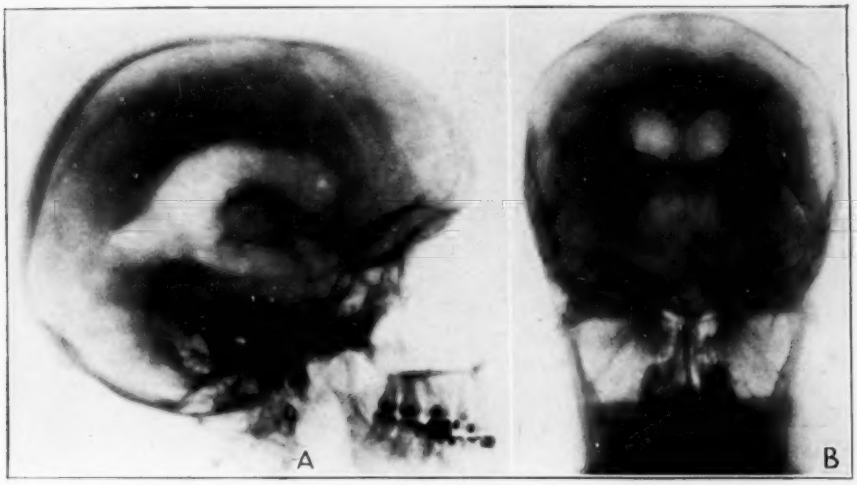

Fig. 3 (case 9).—*A*, lateral view of encephalogram. The lateral ventricle is strikingly dilated, and there is a relatively moderate accumulation of air in the frontal and temporal regions. The sulci are not well marked. Internal hydrocephalus is present with the possibility of leptomeningeal adhesions. *B*, anteroposterior view of the same encephalogram. Note the marked dilatation of both lateral ventricles.

Case 10.—F. B., aged 36, had had meningitis with convulsions at the age of 9; since then, attacks have occurred during the menstrual periods, and in the last few years with greater frequency. There were no neurologic signs. Mentally, she was in an advanced state of deterioration, with frequent episodes of excitement. Encephalography was done on Aug. 1, 1929; only 90 cc. of fluid was obtained, and the same volume of air was injected.

Encephalography showed a large accumulation of air in the frontal, temporal and occipital regions, and an unusually large accumulation in the area of the main pacchionian bodies. The right lateral ventricle was extremely dilated (fig. 4*A*) The sulci were well outlined, and all the cisternae were well filled. The third and fourth ventricles were well outlined. There was pronounced asymmetry in the size of the ventricles, which is seen better in the postero-anterior view, the right

ventricle being twice as large as the left (fig. 4*B*). There was air in the region of the falx. After twenty-four hours a considerable amount of air was still present.

The patient died following a severe convulsion on Sept. 5, 1929, a month and four days after the encephalography, from which she had recovered in a few days. Autopsy was not performed.

Case 11.—J. M., aged 36, fell and struck her head at the age of 7, but showed no sequelae. At the age of 15 she began to vomit once or twice a day at intervals; this continued for about two years. At 17, she began to have convulsive seizures. Except for dilated pupils and a tremor of the fingers, there were no neurologic signs. She was deteriorated and had frequent episodes of excitement. Encephalography was done on Aug. 15, 1929, at which time 145 cc. of fluid was replaced by an equal volume of air.

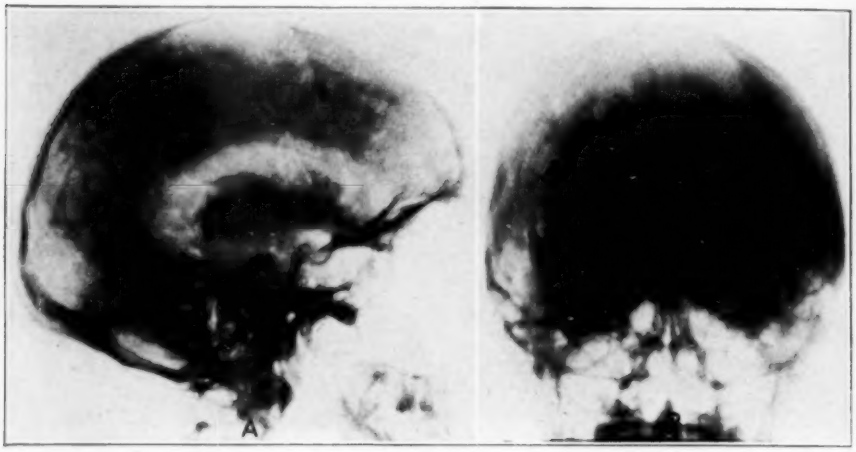

Fig. 4 (case 10).—*A*, lateral view of an encephalogram. The right lateral ventricle is extremely dilated, and there is a large accumulation of air in the frontal and temporal regions and in the area of the main pacchionian bodies. The cisternae are well filled. Internal hydrocephalus is more pronounced on the right side. *B*, postero-anterior view of the same encephalogram. There is pronounced asymmetry in the size of the lateral ventricles, the right being larger than the left, and a large accumulation of air in the vortex. Air can be seen in the region of the falx and there is evidence of more air in the left temporal region on the side of the smaller lateral ventricle.

Encephalography showed that there was a considerable accumulation of air in the frontal and temporal regions, which was better seen in the postero-anterior view (fig. 5*B*). There was a small accumulation of air in the area of the pacchionian bodies, the sulci were well seen except in the parietal region (fig. 5*A*), which may have been due to adhesions of the leptomeninges. The lateral ventricles were much dilated, and the cisternae well filled. After twenty-four hours, practically the same amount of air was still present.

Case 12.—H. D., aged 24, had had acute articular rheumatism with cardiac complications at the age of 9. Generalized convulsions had occurred since the age

of 14. Except for dilated, unequal pupils, a tremor of the fingers and hyperreflexia, there were no definite neurologic signs. The patient had deteriorated from the early years of the illness, although she had improved surprisingly in the past four years under massive doses of bromide and phenobarbital. Encephalography was done on June 3, 1929, and 130 cc. of fluid was replaced by air.

Even the encephalograms taken twenty-four hours after encephalography showed a considerable accumulation of air in the subarachnoid spaces over the frontal, temporal and occipital regions, and also in the area of the main pacchionian bodies (fig. 9*A*). The sulci were well seen. The lateral ventricles were much dilated; the cisternae were unusually large. On the whole, there was a pronounced dilatation of the ventricles, with considerable increase in the size of the subarachnoid spaces. A considerable amount of air was still present after 120 hours and traces even after 216 hours (fig. 9*B* and *C*).

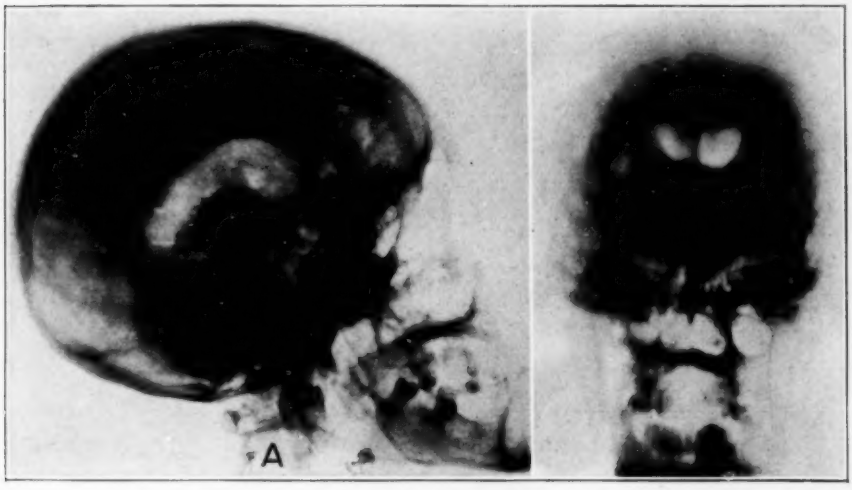

Fig. 5 (case 11).—*A*, lateral view of encephalogram. The lateral ventricle is much dilated and the cisternae are well filled. There is considerable accumulation of air in the frontal region and a small accumulation of air in the area of the pacchionian bodies. The sulci in the parietal region are not well seen. Internal hydrocephalus was present and the possibility of some leptomeningeal adhesions in the parietal region. *B*, postero-anterior view of the same encephalogram. Both lateral ventricles are dilated and are practically symmetrical in size and position. There is considerable accumulation of air in the temporal regions.

Summary and Comment.—The patients in group 2 were somewhat younger than those in group 1, and, therefore, the possibility of senile atrophy of the brain, which may give a similar picture can be excluded with certainty. It is interesting that the degree of dilatation of the ventricles does not seem to correspond to the amount of fluid withdrawn. The average amount of air injected in this group was not higher than that in group 1. In only one patient (case 11) was 145 cc. of air

injected. On the other hand, in case 10, which showed the most characteristic observations of the group, only 90 cc. of fluid was withdrawn and replaced with air. Naturally, the absolute size of the brain in each case and the general size of the patient must be considered in comparative studies of this kind. The mental state of the patients in this group was that of deterioration. This, however, has no especial relation to the specific observations in the roentgenograms, for it will be noted that there were patients in an advanced state of mental deterioration who gave entirely normal encephalographic appearances.

Another interesting observation in this group is the persistence of air for as long as 216 hours. This did not seem to depend on the amount of air injected but rather on the degree of dilatation of the ventricles, as there were patients in group 1 in whom a larger amount of air was injected, with only traces of air after 24 hours. One may assume a circulatory disturbance due to the dilatation of the ventricles and the enlarged subarachnoid spaces, or due to the possible meningeal adhesions which prevent the absorption of air.

Group 3: Miscellaneous Cases with Possible Pathologic Changes.—In this group of five cases I have included patients in whose encephalograms no traces of ventricles could be seen or ventricles that were distorted and asymmetrical both in size and location. I have also included patients with pronounced adhesions of the leptomeninges.

Case 13.—M. D., aged 49, had had convulsions since the age of 16. There were no neurologic signs except for a pronounced difference of color in the two sides of the face. Mentally, the patient was deteriorated. Encephalography was done on April 27, 1929, and 125 cc. of fluid was withdrawn and replaced by an equal volume of air.

Encephalography showed a small accumulation of air in the frontal and temporal regions; no air was visible in the area of the main pacchionian bodies; the sulci were not seen. The lateral ventricles were somewhat dilated; the third and fourth ventricles were seen. There seemed to be adhesions of the leptomeninges all over the surface of both hemispheres as the sulci were not seen.

Case 14.—S. H., aged 33, had had a number of the diseases of childhood, including scarlet fever, measles and whooping cough. At 11, she had typhoid fever. Intellectually, she was retarded to the degree of an imbecile. She began to have convulsions at the age of 24, characterized by generalized rigidity and occasional clonic twitchings in various groups of muscles during the attack. There were no neurologic signs. Mentally she was deteriorated. Encephalography was done on July 18, 1929; only 80 cc. of fluid was obtained and replaced by an equal volume of air.

Encephalography showed a moderate accumulation of air in the frontal and occipital regions, where some of the sulci were seen. Over the rest of the cortex only an occasional patch of air was discernible, giving the impression of extensive adhesions of the leptomeninges. Only a trace of the right ventricle was seen in the lateral and other views. The left ventricle was totally absent. Slight residuals of air were still present after twenty-four hours.

CASE 15.—H. L., aged 43, had had convulsive seizures since the age of 27. She had used alcohol excessively since the age of 34. There were no neurologic signs. Mentally, she was in a state of profound deterioration. Encephalography was done on Aug. 23, 1929, and 155 cc. of fluid was replaced by an equal volume of air.

Encephalography showed an accumulation of air in the frontal and temporal regions which was seen best in the anteroposterior view (fig. 6*B*). There was also an accumulation of air in the occipital region, but little air in the area of the pacchionian bodies. No traces of ventricles were observed in the lateral (fig. 6*A*) and anteroposterior views. The sulci were well seen all over both hemispheres. The cisternae were not well discernible. As there was considerable air in the subarachnoid spaces one must assume that no air had escaped during the injection, and the absence of the ventricular shadows was probably due to the occlusion of the foramens of Monro. After twenty-four hours the air had practically disappeared.

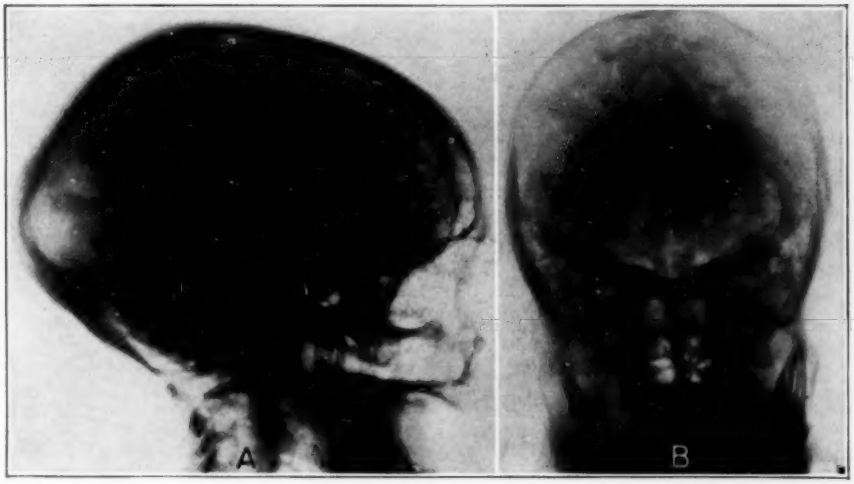

Fig. 6 (case 15).—*A*, lateral view of encephalogram. There are no traces of ventricles, but there is an accumulation of air all over the hemisphere, yet relatively little in the area of the pacchionian bodies. *B*, anteroposterior view of the same encephalogram. There are no traces of the lateral ventricles. Note the considerable accumulation of air over both hemispheres and the air in the region of the falx.

The patient died on Oct. 27, 1929, two months after encephalography was performed. Death was due to a sudden hemoptysis. Autopsy was refused.

CASE 16.—I. H., aged 23, who had had numerous diseases in childhood, had early shown evidence of mental deficiency. Convulsions had occurred since the age of 7. There were unequal pupils, a tremor of the fingers and hyperreflexia. There was a progressive and profound deterioration. Encephalography was performed on Aug. 23, 1929; only 75 cc. of fluid was withdrawn and replaced by air.

Encephalography showed a moderate accumulation of air in the frontal and temporal regions (fig. 7*A*), and a patch of air in the region of the pacchionian

bodies. The sulci in places were well marked; in others they were not seen. The left ventricle in the lateral view was somewhat dilated, considering the small encephalon. In the postero-anterior view (fig. 7*B*) there was a pronounced asymmetry of the ventricles. The left was almost three times as large as the right. After twenty-four hours the right ventricle was not seen at all, while the left was well filled with air.

Case 17.—B. M., aged 18, had had convulsions since the age of 13. Since that age, athetotic and choreic movements had occurred in all extremities so that dystonia musculorum deformans had been considered. The movements had entirely disappeared in the year and a half before consultation; the convulsive attacks, however, were still present and were characteristic, consisting of rigidity resembling opisthotonos and attitudes of the hands reminding one of a picture of decerebrate rigidity. The pupils were unequal; there were pronounced hyper-

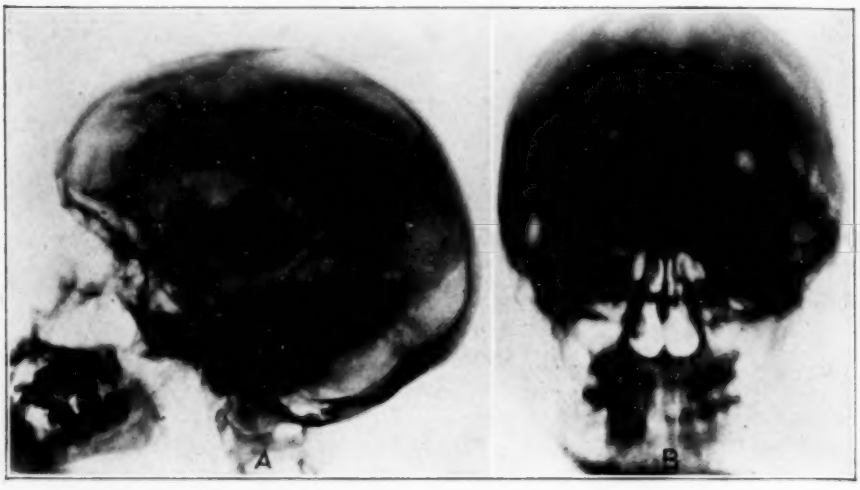

Fig. 7 (case 16).—*A*, lateral view of encephalogram. There is a moderate accumulation of air in the frontal and temporal regions and a patch of air in the area of the pacchionian bodies. The sulci in places are not well marked. The right ventricle is somewhat dilated. *B*, postero-anterior view of the encephalogram. There is an accumulation of air in the area of the main pacchionian bodies and pronounced asymmetry of the lateral ventricles, the left being three times larger than the right.

reflexia and vasomotor instability. Emotionally, the patient was an unstable psychopath; intellectually, she was normal. Encephalography was done on June 20, 1929, and 140 cc. of fluid was withdrawn and replaced by an equal volume of air.

Encephalography showed considerable accumulation of air in the frontal and occipital regions, but no air in the area of the pacchionian bodies. The sulci were well marked all over the hemispheres. The lateral ventricles were extremely narrow and distorted.

Summary.—This group of five cases seems to present a varied pathology. In case 13, for instance, one would expect to find extensive adhesions of the leptomeninges. In case 15 there is probably occlusion of the foramens of Monro, and in case 17 some process which has narrowed and distorted the lateral ventricles. In practically all of the patients there was a history of numerous illnesses in childhood which may have produced an encephalitic or meningo-encephalitic process.

GENERAL COMMENT

In order to exclude mistakes in the interpretation of the encephalograms, I made a number of roentgenograms immediately before the air was injected. In some of these, there are shadows in the frontal

Fig. 8 (case 10).—*A*, lateral view of roentgenogram taken before encephalography. Compare with figure 4*A*. Note the shadow in the area of the main pacchionian bodies in the roentgenogram while there are no shadows elsewhere.

and occipital regions which in the encephalograms might pass for accumulations of air. However, these shadows are not always present; when they are, they are not so pronounced as in the encephalograms. In case 10 (fig. 4*A*), I found a definite shadow in the area of the pacchionian bodies in the encephalogram and in a roentgenogram that was taken before the injection of air (fig. 8). No shadows are to be seen in this roentgenogram in the frontal or occipital regions, so that one may consider the shadow in the area of the pacchionian bodies as evidence of a definite dilatation of the subarachnoid spaces in that region. I am therefore inclined to believe, in view of the fact that shadows are present only in a limited number of roentgenograms taken

before the injection of air, that they may indicate an accumulation of subarachnoid fluid discernible without the help of encephalography. I realize the complicated factors of physics involved in this problem, and offer this explanation tentatively only.

Air was still present in the ventricles and subarachnoid spaces after twenty-four hours. In case 9, the ventricles appear even better filled

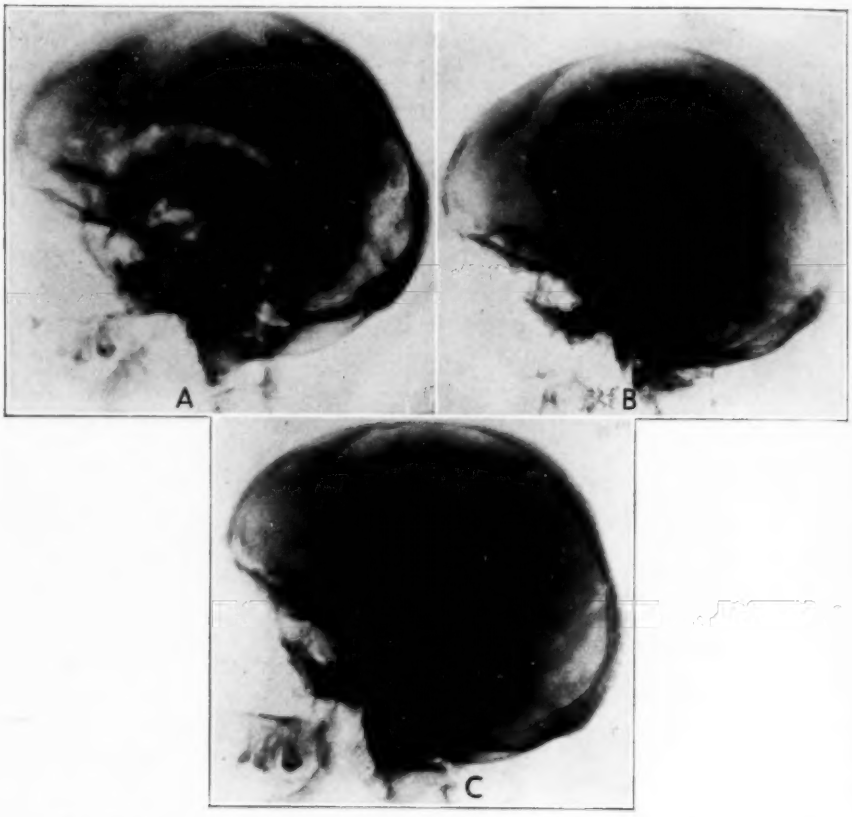

Fig. 9 (case 12).—*A*, lateral view of encephalogram taken 24 hours after encephalography. *B*, lateral view taken 120 hours after encephalography. Note the well outlined lateral ventricle with all the horns discernible. *C*, lateral view taken 216 hours after encephalography. Note the presence of air in the lateral ventricle.

and larger in the encephalograms taken twenty-four hours after encephalography, as do those in case 12 (fig. 9*A*). In this patient the air was still present after 120 hours (fig. 9*B*) and even after 216 hours (fig. 9*C*). As was pointed out before, the presence of air after long intervals can be correlated with the degree of dilatation of the

ventricles, but not with the total amount of air injected, as in the case of the patient from whom only 90 cc. of fluid was withdrawn and replaced by an equal volume of air.

I cannot express an opinion here as to the relationship between the frequency of convulsive attacks and the degree of the ventricular dilatation, as all the patients were receiving therapy at the time of the encephalographic procedure with a relatively good response.

No seizures occurred while the encephalograms were being performed or during the following four weeks.

Meningeal effects were observed in all of the patients. Each complained of severe headache for forty-eight hours or longer after the encephalography. In practically every one there was rigidity of the neck, and all showed profuse perspiration, vomiting and acceleration of the pulse rate, followed by pronounced bradycardia. The latter signs cannot be considered as related only to the encephalographic procedure; for many other factors must be taken into consideration, such as the sedatives administered in preparation for the encephalography and the cardiac stimulants frequently given during the procedure and afterward. I think, however, that the meningeal signs can be reduced if a volume of air is injected less than the amount of fluid drained, because air expands under the higher body temperature and thus adds to the meningeal irritation.

CONCLUSION

Encephalographic studies have been made on a group of seventeen patients with recurrent convulsions of unknown cause ("idiopathic epilepsy"). None of the patients showed clinical evidence of a focal lesion. The selection was made purposely, as the study has been made with the aim of ascertaining possible pathologic changes in the idiopathic group.

The ages of the patients ranged between 18 and 49 years. Three showed no evidence of mental deterioration, one was mildly deteriorated, while the rest were in a more or less advanced state of deterioration.

In eight patients, or 47 per cent, the observations were normal; in four, or 24 per cent, dilatation of the ventricles and of the subarachnoid spaces and some evidence of leptomeningeal adhesions were found. In five other cases, or 29 per cent, the encephalographic observations seemed to point to a definite pathologic process, inflammatory or perhaps neoplastic, but apparently not active. No correlation can be made between the degree of the deterioration and the extent of the pathologic changes.

Air was still present after 24 hours in all cases, and sometimes after 216 hours. Its presence seems to correlate more with the extent of the

dilatation of the ventricles and of the subarachnoid spaces than with the amount of air injected.

Subarachnoid dilatations may rarely be seen in roentgenograms taken before the injection of air.

A comparative study of roentgenograms made before and after the injection of air is desirable in order to exclude the possibility of faulty interpretations of the encephalograms.

INDUCTION OF SLEEP IN EPILEPTIC PERSONS*

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In the course of experiments on the induction of sleep in nonepileptic persons by means of an apparatus described in a paper previously published,[1] it appeared that the induced sleep was unlike ordinary sleep in that it was, so far as could be observed, largely devoid of phenomena of release. For instance, it is a common observation that when a person lies down to sleep he very rarely falls asleep in the position in which he first lay down. He usually turns one or more times, flexes and extends his body and limbs, and falls asleep in a given habitual posture. If one tries, by way of experiment, to fall asleep flat on the back with the body and limbs extended, unless such a posture is habitual, one will be convinced of the difficulty of the attempt as well as of the important part that phenomena of release play in the onset of sleep. In thousands of observations on the sleep induced by means of the mechanical appliance discussed in this paper, there was not a single instance of a person's wishing to turn from the original position—flat on the back and with the body and limbs extended—in which he was placed in the apparatus.

The following facts were observed: 1. If a person in this apparatus was rudely awakened in the first stages of sleep, he was, on the average, startled much less than persons who are disturbed in the onset of ordinary sleep. 2. In the numerous experiments the spontaneous startled movements, which are not infrequent during the beginning of ordinary sleep,[2] never appeared in the induced sleep. 3. The absence of true dreams was observed. Although in the course of disintegration of the sensory function which takes place during the onset of sleep the person may have experienced a confusion and incoherence of his line of thought, a reexperience of past sensory experiences, which constitutes a true dream, never occurred. 4. Muscular relaxation took place from the beginning of the action of the apparatus and was simultaneous with the experience of a sense of somnolence.

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* Submitted for publication, Sept. 22, 1930.

* From the Department of Neurology, Columbia University.

* Read at a meeting of the Association for Research in Nervous and Mental Disease, New York, December, 1929.

1. Rosett, Joshua: An Apparatus for the Induction of Muscular Relaxation and Sleep, Arch. Neurol. & Psychiat. **22**:737 (Oct.) 1929.

2. Gowers, William R.: The Borderland of Epilepsy, Philadelphia, P. Blakiston's Son & Company, 1907, p. 106.

A fifth observation, pertinent to the conclusion of an absence of phenomena of release in the induced sleep, is of a more complicated nature than the preceding ones. Many persons can gage, without gross error, the length of time during which they have been asleep, and certain persons can awaken at an appointed time in spite of the habit of sleeping beyond that time. This phenomenon is usually explained by the reasonable assumption that people learn to a certain extent to measure duration of time in their sleep by the succession of functions within the body. In the sleep induced by means of the apparatus to be described, all persons erred grossly regarding the duration of the sleep, always underestimating it and never overestimating it. A large number of persons who were convinced that they merely "dozed off" for a minute or two, had really been asleep for as long as an hour; and the same persons repeated the same error over and over. Training, in this respect, appeared to be utterly without avail. In a large number of instances, indeed, the numerical expression of the error amounted to infinity, that is, the person was convinced that he did not sleep at all, when as a matter of fact close observation showed him to be soundly asleep. Therefore, the number and intensity of events in the body by means of which sleeping persons are more or less enabled to gage time must have been very much diminished. That the events in the body which consist of the activity of the voluntary musculature were reduced in number and intensity was evident early in the experiments. A little later, it appeared that the contraction of the involuntary musculature was diminished as well. This was evidenced by the flushing of the skin and by a fall in the blood pressure. However, it is only within the last few months that the fact of a diminution of the activity of certain mechanisms of large scope which subserve muscular contractions was ascertained. I owe the knowledge of these facts to the studies of Dr. F. T. Farnell of Providence, Rhode Island, and of Dr. Leon Cornwall of New York, who severally communicated to me the results of their work now in progress. Dr. Farnell made estimations of the carbon dioxide combining power of the blood and found uniformly a drop of from 2 to 10 points immediately after the induced sleep. Dr. Cornwall measured the quantity of sugar in the blood and found a decided reduction immediately after treatment with the apparatus. On the basis of a general diminution of the number and intensity of events in the bodies of persons subjected to the action of the apparatus in question, the foregoing observations largely explain the remarkable defect in the faculty of estimating time.

A review of the physiologic action of sedatives, narcotics and anesthetics reveals that, although the end-results of all of these drugs consist, on the whole, in general anesthesia and muscular flaccidity of varying degrees of intensity, they exhibit certain essential differences dur-

ing the progress of their action, according to which they may be divided into two groups. Drugs represented by morphine, cannabis indica, alcohol, chloroform and ether constitute one group; those represented by the bromides and by phenobarbital constitute another. Although both groups produce a diminution or blocking of the conduction by the nerve, the clinical manifestations of the action of the first group are indicative of a successive disabling of the several neurons of the long or cerebral arc along the direction of its conduction; those of the second, of a simultaneous involvement of all the neurons of that reflex arc.

Although it is not easy for a neurologist who thinks in terms of nerve pathways to decipher the argument of the pharmacologist who speaks of the effect of drugs on nerve centers, a few quotations may still be enlightening. It will be seen that in the mosaic of statements regarding the action of these drugs, there is a pattern that separates them into two distinct groups. The following citations are typical.

In discussing the action of alcohol, belladonna, stamonium, hyoscine or scopolamine and cannabis indica (p. 407), Rusby, Bliss and Ballard [3] defined them as:

> Medicines capable of producing a stupor somewhat resembling sleep, following a state of delirium. The delirium is the result of depression of the powers of judgment and control. In this action, therefore, the narcotics are cerebral depressants. The prominent symptoms of the first stage are mental and usually also physical activity, talkativeness of a kind that shows want of proper restraint . . . and probably, the most significant of all, depression or blunting of the sensory centers. . . . The increased activity, due to decreased control, is followed . . . by depression of all functions which soon lose their activity altogether. Depression of sensation increases, thus leading into the second stage of dulness and quietude and finally into stupor. . . .

In their discussion of the bromides (p. 428), they said:

> The action of these compounds may be summarized in the statement that they are general depressants of the nervous system. Perhaps their tendency to reduce motor functions is secondary to sensory depression, with consequent diminished reflexes, but it appears that in part, at least, the motor areas are directly affected.

In reference to the general action of alcohol, ether, chloroform and allied drugs, Torald Sollmann [4] (p. 707) said:

> The main actions [of these drugs] are similar; but they show practically important differences in severity, rapidity, duration and sequels. The successive effects may be divided in stages . . . (1) analgesia; (2) light anesthesia; (3) deep anesthesia; (4) bulbar paralysis. . . . The reflexes are at first rather exaggerated, later somewhat depressed. . . . The sensibility is impaired. . . .

3. Rusby; Bliss, and Ballard: The Properties and Uses of Drugs, Philadelphia, P. Blakiston's Son & Company, 1930.

4. Sollmann, Torald: A Manual of Pharmacology, Philadelphia, W. B. Saunders Company, 1928.

The thoughts become confused, with hallucinations, sensation of stiffness and want of muscular control. [In the succeeding stage] consciousness, sensation and most reflexes are lost . . . consequently the muscles relax.

Concerning opium, morphine and related alkaloids (pp. 286 to 287) Sollmann said:

The first effect produced by doses too small to elicit any other symptoms consists in diminished sensibility. . . . Somewhat larger doses depress the attention and thus weaken the appreciation of other external impressions. . . . Some individuals . . . are apparently more excited than depressed by morphine. This can perhaps be explained by diminished restraint, rather than by direct stimulation. The same explanation probably holds true also for the flights of imagination which fill the period producing sleep. . . . Codeine and diacetylmorphine are less quieting and perhaps more excitant and convulsant than morphine.

Of the bromides (p. 878) Sollmann said:

A single dose of 1 to 2 Gm. of bromides has no noticeable action on normal individuals. Above 4 Gm., depression and decrease of reflexes become apparent. The effects are rather different from those of ordinary hypnotics: The bromides tend to produce a mental calm . . . progressing to sluggishness, lack of attention and lassitude. These all dispose toward sleep. . . .

Of the barbital series of drugs (p. 757) Sollmann said:

The usual dose induces a deep, dreamless sleep.

Cushny,[5] writing on cannabis indica (pp. 263-264), said:

The effects of cannabis indica are chiefly due to the changes in the central nervous system, in which it induces a mixture of depression and stimulation similar to that seen occasionally under morphine. . . . Soon after its administration, the patient passes into a dreamy, semi-conscious state, in which the judgment seems to be lost, while the imagination is untrammeled by its usual restraints. The dreams assume the vividness of visions, are of boundless extravagance, and, of course, vary with the character and pursuits of the individual. . . . Ideas flash through the mind without apparent continuity, and all measurement of time and space is lost. True hallucinations may appear. . . . The sensation of pain is lessened or entirely absent, and the sense of touch is less acute than normally. Later the dreams alternate with periods of complete unconsciousness, from which the patient can be aroused easily, and the symptoms eventually pass into tranquil sleep.

Speaking of veronal (barbital) (p. 236), Cushny stated:

Veronal, diethylbarbituric acid, and its sodium salt, medinal, in ordinary doses induce natural sleep and larger quantities deepen and lengthen the unconsciousness without other organs than the central nervous system being involved, though the patient may complain of lethargy and drowsiness subsequently.

5. Cushny, Arthur R.: A Text-Book of Pharmacology and Therapeutics on the Action of Drugs in Health and Disease, ed. 7, Philadelphia, Lea & Febiger, 1918.

With regard to bromides (p. 265), Cushny wrote:

The first symptom is often a dull, heavy headache, with a feeling of lassitude, fatigue, disinclination for exertion, mental or physical, and often muscular weakness. Thought is slow and confused, the memory is indistinct, ideas are put into words with difficulty and the speech is accordingly slow and hesitating. External objects and movements are perceived, but arouse no interest in the patient, and very often this state of apathy passes into drowsiness and sleep.

The most lucid account of the action of these drugs is found in a book published thirty years ago. White and Wilcox,[6] writing on alcohol (pp. 268 to 269), said:

The highest functions are most affected. The person who has taken the alcohol talks more fluently and brilliantly, his wits are sharpened, he has a feeling of strength. If the dose has been large, the stage of exaltation of these or any other functions quickly passes into one of depression, the highest functions being affected first, and the stimulation and depression of function proceed regularly from the highest to the lowest. The action of alcohol thus illustrates the "law of dissolution," which states that functions which have appeared latest in the animal series or the individual are the most easy to influence, those which have appeared earlier are less easy to influence; and so by regular sequence till we arrive at those functions which are first developed, which are the last to be influenced. . . . The power of judgment is abolished very early by alcohol; this is so while the imagination, the emotions, and the power of speech still remain stimulated; but soon the power of imagination goes, the patient loses all command over his emotions, he cries and laughs irregularly, but this soon stops. He next begins to lose control over his speech, talking incoherently and thickly; shortly afterwards he cannot talk at all, but can only make a noise. Muscular movements, which are not so highly developed as those of speech, are next affected; delicate, lately developed movements, as writing, feeding himself, etc., are for a time performed inco-ordinately, but soon they are paralyzed. Next the muscular movements, developed before these, are implicated, and the patient cannot undress himself or walk straight, and inco-ordination of these movements passes into the inability to do them at all. Next the activity of the reflex centres of the cord is abolished, the patient passes his urine and feces (involuntarily).

With regard to chloroform (pp. 275 to 276), these authors said:

First stage. The stimulation of all higher functions is very transitory, and he [the patient] quickly begins to lose consciousness; he may be aware that people around him are talking without knowing what they are saying, but soon he hears and sees nothing. Sometimes during the early part of this state he may laugh or cry. The inability to see and hear is quickly followed by considerable blunting of general sensation. At the same time that these higher functions are being depressed the lower motor functions are excited; he will kick and fight, throwing his arms and legs about, so that much strength may be required to hold him down, and he will shout and talk incoherent nonsense very loudly.

Second stage. He sees, hears and feels nothing, hence chloroform is called a *general anesthetic*. The excitement of the motor functions passes into depression, and he ceases to shout and struggle.

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6. White, Hale W., and Wilcox, Reynold W.: Materia Medica, Pharmacy, Pharmacology and Therapeutics, American ed. 4, Philadelphia, P. Blakiston's Son & Company, 1898.

On the action of opium (pp. 332 to 333) White and Wilcox wrote:

> The higher faculties are at first excited even by small doses. In a few persons there is no inco-ordination in this excitement. The intellectual power and mental vigor are increased, and therefore the drug is taken by some people to enable them to do their mental work. Usually, however, the excitation does not affect the mind evenly; generally the imagination is powerfully and pleasantly excited, much more so than the faculties of reason and judgment, which are a little dulled. . . . This is soon succeeded by *sleep,* which is accompanied by pleasant dreams, generally of an impossible nature. . . . This, which is the beginning of the depression of the highest centres, is soon followed by depression of the others, the higher being influenced before the lower, so that soon the sleeper does not respond to any sound, light, or cutaneous stimulation, *nor does he feel pain.* . . . If a large amount is given, often there is no primary excitement, and then the first symptom that opium has been taken is drowsiness.
>
> The motor cells of the *spinal cord* are at first slightly stimulated, and consequently reflex excitability is exaggerated; but they are soon *depressed,* and it is difficult to obtain reflex movements.

Speaking of bromides (pp. 238 to 239) White and Wilcox wrote:

> Bromides are powerful *depressants to the nervous system;* (the action of the potassium salt being most marked). Thus, if an animal be given large doses of any of them, irritation of the cortical motor areas, which before easily excited movements, fails to do so. Experiments also show that the reflex excitability of the cord is considerably diminished, and that the activity of the sensory mechanism is also impaired, for large doses of bromides given to frogs cause cutaneous anaesthesia. In man, at least, not only the cortical motor area, but the brain as a whole is depressed, therefore these drugs are powerful *hypnotics.* It is probable that in addition to the brain and spinal cord the peripheral nerves are depressed, so that bromides are well worthy to be called powerful nervous depressants. The activity of the muscles is also diminished, not only by the action of the drugs on the nervous system, but by their direct action on them.

Therefore, the clinical manifestations of the action of the drugs of the first group are qualitatively different during different stages of their action. The first stage, which is manifested by a certain degree of disorientation, that is, a defective appreciation of and response to the environment, is generally coincident with an increased experience of the emotions. As the only symptom produced by a disabled nerve structure is an absence of its inherent function, the syndrome of disorientation that is coincident with an increased experience of emotion might be attributed, in the present state of knowledge, to an interruption of the cerebral reflex arc at a point between the optic thalamus and the cerebral cortex. The reception of impressions from the environment is therefore diminished, while the function of the thalamic mechanism is released and activated. The second stage is characterized by increased disorientation; and this manifestation of a defect of the sensory function is frequently coincident at first with simple hallucination and later with composite

ones. Such a clinical syndrome may be interpreted as an interruption of the cerebral reflex arc at points in the cerebral cortex that mark the beginning of the association systems, the function of which is therefore released and activated. The further action of these drugs is marked by deepening unconsciousness and a certain degree of muscular rigidity and abnormal movement, probably indicating a disability of the cerebral association system as far as the beginning of the long efferent systems. This stage merges into one of muscular flaccidity which is interpretable as a disability of the entire reflex arc.

Since an epileptic attack is likewise marked by the series of symptoms outlined, one can see the rationale of the injurious effects of these drugs on epileptic persons.

In contrast to the action of the drugs mentioned is that of the bromides and of phenobarbital. The manifestations produced by these drugs is, from the very beginning of their action, a diminution of the sensory function, peripheral as well as central, that is, diminished receptivity as well as a diminution of the power of imagery, thought and projection; simultaneous with this sensory defect is a diminished activity of the musculature. The succeeding stages of the action of these drugs are marked not by qualitative but merely by quantitative differences. The sensory defect and the muscular relaxation continue to increase until the onset of sleep, which is marked by thorough muscular relaxation and is devoid of hallucinations. Such an absence of symptoms of release and activation during the progress of general neural disability can best be explained by assuming that, in its progress, the disability is at any moment equally distributed among all the neurons that constitute the long reflex arc.

Therefore, it appears that the beneficial action of the bromides and of phenobarbital in epilepsy is due not merely to the fact that they are sedative and hypnotic drugs, but to the fact that, by affecting all the neurons of the reflex arc simultaneously, they counteract those phenomena of release and activation in the different portions of the reflex arc which bring about the series of events of the epileptic seizure. That such is the case will be presently evidenced by the fact that a purely mechanical apparatus, the effect of which is a diminution of nerve conduction simultaneously throughout the nervous system and which is therefore devoid of release phenomena, is productive of the same results in epilepsy as the administration of the bromides and of phenobarbital. Like these drugs the measure in question is but palliative of the symptoms and is not productive of permanent effects.

It will be observed that the action of the first class of drugs is analogous to that of the onset and progress of ordinary sleep, and this analogy becomes more apparent when it is remembered how favorable is the onset of ordinary sleep to the occurrence of the epileptic seizure, while the action of the second class of drugs is like the onset of that

exceptional sleep which is devoid of dreams and of startled movements and is marked from the beginning by muscular relaxation.

These facts and their theoretical explanation made the conclusion seem reasonable that the action of the apparatus on the nervous system is similar to that of the bromides and phenobarbital. With but little apprehension of having the apparatus damaged by a possible convulsion, experiments on epileptic persons were begun.

At the date of writing, over 1,000 treatments had been administered to fourteen epileptic persons. The patients were chosen with a view to the chronicity, severity and variety of symptoms, as will appear from the report of some of the cases. The attacks were both nocturnal and diurnal. All of the patients had been taking drugs for years. They were placed on reduced doses of phenobarbital and were carefully observed during the treatments. With the exception of one patient, who will be mentioned, the patients fell asleep after the first few treatments in from one to three minutes from the beginning of the action of the apparatus; children succumbed to its action more readily than adults. After each treatment they remained relaxed for many hours like non-epileptic persons, and they slept well on the following night. In all the hours of sleep not a single epileptic seizure had taken place during the treatment. The general effect on the seizures will be gathered from the following case reports.

REPORT OF CASES

Case 1.—S. M., a man, aged 42, had been afflicted for about a year with grinding his teeth and biting his tongue almost every night. Five grains (0.324 Gm.) of phenobarbital, taken at night for some months, had brought no relief. When first seen, he complained of great loss of strength, which he attributed to inability to sleep at night for fear of biting his tongue.

The drug was discontinued, and thirty-four treatments by means of the apparatus were administered within ten weeks. During the first few treatments he was observed to grind his teeth slightly, but he did not bite his tongue. During the first month of treatment he bit his tongue at night four times, and once about the middle of the second month. It was noted that the five attacks took place on nights following days on which he received no treatment. He ceased biting his tongue about the middle of the second month, and ceased grinding his teeth at the end of eight weeks.

Case 2.—G. H., a woman, aged 25, had had severe major attacks on an average of three or four a week for four years. The administration of 15 grains (0.972 Gm.) of phenobarbital a day reduced the number of attacks to three or four a month. In the course of fifteen months, 338 treatments were administered. The patient slept so soundly in the apparatus that she had to be awakened each time after the treatment. The dose of the drug was reduced to 4½ grains (0.292 Gm.) a day for the first six months of this period. During the first three months she had eight very mild major seizures and five of a minor nature during the next three months. The dose of phenobarbital was reduced to 1½ grains (0.087 Gm.) a day, and during the next six months she had one mild major attack. During the next month of treatment she took daily ¾ grain (0.048 Gm.) of phenobarbital and had one very minor attack. In the last six weeks, medication was discontinued

and she had one mild major seizure. The report of the parents is that the character of the girl has shown a decided change for the better. She is no longer impulsive, pugnacious and discontented, but deliberate, gentle, helpful and happy.

Subsequent Note.—With respect to permanency, the efficacy of this treatment is no greater than that of the drugs usually employed in epilepsy; this is illustrated by the subsequent history in this case. The treatment was not administered during the months of July and August, 1930. In spite of taking phenobarbital (doses uncertain), the patient had twelve major attacks during those months.

Case 3.—S. C., a man, aged 56, had been afflicted almost daily for nineteen years with one major and one minor attack. He had been taking bromides and phenobarbital for years in doses which I could not ascertain. He was given 3 grains (0.195 Gm.) of phenobarbital a day, and 226 treatments by means of the apparatus were administered in the course of fifteen months. During the first ten days, he had three major and four minor attacks. No major attacks and seventy-one minor ones, consisting of a sense of dizziness, occurred in the next twenty-two weeks—an average of one in two days. After the twenty-second week, the dose of the drug was reduced to 1½ grains a day. For the last six months he had had no major and fifteen minor attacks.

Case 4.—J. H., a man, aged 39, had had one major and two minor attacks almost daily for over three years. He had been taking phenobarbital and bromides in doses the amounts of which I could not ascertain. He was given 1½ grains of phenobarbital a day, and sixty-six treatments by means of the apparatus were administered in the course of eleven weeks. During the first four weeks, he had ten major attacks and twenty-four minor ones. During the next four weeks, he had no major attacks and sixteen minor ones. The last three weeks he had had one major attack and two minor ones.

Case 5.—T. D., a girl, aged 10, had had major attacks that recurred nightly, frequently with an additional attack during the day, and minor attacks that recurred at least once a day. She had been taking, on an average, 3 grains of phenobarbital a day. The dose of the drug was reduced to 1½ grains a day, and twenty-six treatments were administered by means of the apparatus in the course of five weeks. She had five major and six minor attacks during the first week; nine major and five minor ones during the second week; five major and seven minor in the third week; one major and two minor in the fourth week, and two minor attacks in the fifth week.

Case 6.—S. F., a man, aged 35, had been having four mild major attacks a week, and minor attacks, consisting of a sense of dizziness, almost daily for fourteen years. He had been taking 1½ grains of phenobarbital a day; this was continued during the treatment by the pneumatic apparatus. The results of fifteen treatments administered within three weeks was as follows: in the first week he had two major and two minor attacks; in the second week he had one major and six minor attacks, and in the third week six major and three minor attacks. The patient became discouraged with the ineffectiveness of the treatment and had not returned.

Case 7.—A. S., a man, aged 26, had been having violent major attacks once every month and a minor attack every other day for six years. He suffered, besides, from a variety of mild mental conditions—fears, anxieties and vague apprehensions. He had been taking phenobarbital at irregular intervals in doses the amounts of which I could not ascertain. In the first three months of treatment by means of the apparatus, no drugs were given; in the next two months 1½ grains of phenobarbital; in the fifth month the patient complained more than

usually of the mental symptoms, and 45 grains (2.92 Gm.) of bromides were given daily and 1½ grains of phenobarbital every other day. In the course of the five months, 135 treatments were administered. During most of the treatments he dozed rather than slept in the apparatus, and a number of times he remained wide awake. In the first month he had twenty-two minor and one major attacks; in the second month, no major and nine minor attacks; in the third month, no major and thirteen minor attacks; in the fourth, no major and nineteen minor attacks, and in the fifth month, no major and twenty-one minor attacks. The patient had preserved a detailed record of the seizures for the past four years. The treatments in question appear to have diminished the frequency and severity of the attacks, but the mental state remained unimproved.

Case 8.—H. R., a girl, aged 11, had had repeated convulsive attacks at the age of 2, lasting a number of hours. After that age, she had mild major and minor attacks—as many as fifteen a week. For the last four years, she had been taking phenobarbital in doses of from ½ grain (0.0324 Gm.) to 1½ grains a day. The nature of the attacks changed; they consisted of the following automatism: She suddenly stopped in the midst of any activity in which she may have been engaged, and began to count on her fingers aloud: "one, two, three, one, two, three," or else she either hummed or sang a song, thanking Mister Grocer or Butcher for what he had brought. The dosage of the drug was established at ½ grain of phenobarbital once a day, and in the course of three weeks, seventeen treatments have been administered. During the three weeks of treatment she has had one mild attack of automatism.

The records of other epileptic persons treated by means of the apparatus presented cases similar to those reported.

SUMMARY AND CONCLUSIONS

Sedative, narcotic and anesthetic drugs may be divided into two distinct groups. The administration of drugs of the first group results in the production of phenomena of release and activation and finally of muscular flaccidity. This may be interpreted as being due to the successive involvement of the several neurons that make up the long or cerebral reflex arc. Administration of drugs of the second group results in an absence of phenomena of release and activation. This may be interpreted as being due to a simultaneous involvement of all of the neurons that make up the long or cerebral reflex arc. The clinical picture of the effect produced by the first group of drugs is similar to that of the epileptic seizure and to the onset of normal sleep. In the same manner that the onset of normal sleep is favorable to the occurrence of the epileptic seizure, so the action of these drugs is rather favorable than otherwise to the occurrence of epileptic seizures. The action of the second class of drugs, which tend to counteract the symptoms of the epileptic seizure, is like that of exceptional sleep that is devoid of release phenomena and that is characterized from the beginning by muscular relaxation. Such sleep can be produced artificially by means of a mechanical appliance. Like the second class of drugs in question, therefore, such sleep has the effect of temporarily diminishing the frequency and severity of epileptic seizures.

MASS RESPONSIVENESS IN THE CENTRAL NERVOUS SYSTEM *

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A review of the literature relevant to the present study discloses certain controversial matters on which our report may throw additional light, notably the concepts of "cerebral localization," inherent rhythm of afferent and on-relaying mechanisms and the genesis of action currents from muscles. The interpretation of the doctrine of cerebral localization of function is changing from a rigid to a more liberal one. The results of present-day work in neurophysiology are steadily attesting to the advisability of such a modification.[1]

Past workers have invested sensory and central neural mechanisms with inherent specificities of response implying only the final motor pathway to be capable of aperiodic rhythm. Here we are unable to find any characteristic rhythm that is not common to every part of the conducting stimulus-response circuit.[2]

To date, the origin of the electromyographically determined action current frequencies is in question. Whether it is resident in the central nervous system or in the muscles or in both has been the point of discussion. Our results would picture the muscle as merely amplifying the action currents—not at all altering their frequencies.

Our earlier experimentation [3] indicated a constant relationship existing in the central nervous system between levels of highest and lowest order of development; the transcortical or highest appeared to be a functional part of the lowest levels of irradiation. These observations

* Submitted for publication, Aug. 28, 1930.

* From the Psychopathic Hospitals, Iowa City and Ann Arbor, Mich.

1. Lashley, K. S.: Brain Mechanisms and Intelligence, Chicago, University of Chicago Press, 1929. Head, H.: Aphasia and Kindred Disorders of Speech, London, Cambridge University Press, 1926. Coghill, G. E.: Anatomy and the Problem of Behavior, New York, The Macmillan Company, 1929.

2. In this we are in agreement with Adrian's observations relative to the sensory component of the conducting circuit.

3. Travis, L. E., and Dorsey, J. M.: Effect of Alcohol on the Patellar Tendon Reflex Time, Arch. Neurol. & Psychiat. **21**:613 (March) 1929; Patellar Tendon Reflex Time in Psychiatric and in Neurologic Cases, ibid. **22**:99 (July) 1929. Travis, L. E., and Herren, R. Yorke: The Relation of Electrical Changes in the Brain to Reflex Activity, J. Comp. Psychol., to be published.

harmonized with a unit-mass envisagement of the central nervous system and furnished direction for further experimentation presented in part in the present study. In it we demonstrate that a one-to-one relationship exists between certain stimulus frequencies and impulse response rhythms in the brain and effector mechanisms. To our goal we were assisted by noteworthy means in the form of a technic which permitted the synchronous recording of both stimulus and response patterns; the sound waves and the action currents were photographed together. So that as natural as possible conditions might obtain in our subject (the dog), his own voice was for the most part used as the stimulus.

METHOD

For recording the action currents, essentially the same apparatus that has been described in previous papers was used. Briefly, it consisted of a three-stage, resistance-coupled amplifier and an oscillograph (Westinghouse). A General Radio low frequency oscillator furnished a time line of either 500 or 1,000 complete cycles per second.

For recording the voice and the whistle we used a Jenkins and Adair C6 condenser microphone with one additional stage of amplification. The output transformer of the extra stage was designed to match the impedance of a supersensitive oscillograph element. All recording was made on Eastman improved orthochromatic motion picture negative.

The action current circuit did not appear to respond to the dog's voice, the Galton whistle or the voice circuit.

The electrodes consisted of two fine silver wires sharpened to needle points and covered with a very thin coat of insulating bakelite varnish. The ends of the needles were bared 2 mm. and coated with silver chloride to prevent polarization. The electrodes were approximately 6 mm. apart for the brain and 4 mm. apart for the cricothyroid muscle.

Throughout the experiment a dog was the subject. Two stimuli were used, the dog's voice and the Galton whistle. The action currents were obtained from the exposed cricothyroid muscle and from both the cerebral cortex and the centrum semiovale. The areas of the brain studied were the frontal (including the so-called sensory and motor zones), the temporal, the parietal and the occipital.

Ether anesthesia was used for all operations. Consecutive records were obtained from the time the dog began to recover from the anesthesia until and during the time his activity reflected a clear comprehension.

RESULTS

Observations on the Cricothyroid Muscle.—Striking synchronization was found between the sound-wave frequency in phonation (whining and barking sounds) and the action current frequency in the cricothyroid muscle (fig. 1). Moreover, a modification in the one was attended by a corresponding modification in the other. The range of these frequencies observed in our records was from 250 to 1,500 per second. It is to be remarked that this synchronization was not always

recorded—rather in no determined regular way there were also recorded action currents which did not synchronize with the sound waves.

Cutting the left first pharyngeal ramus supplying the homolateral cricothyroid muscle helped to isolate near its inception the sensory component of the stimulus-response circuit established by the vibration of the vocal bands (fig. 2). With the motor impulses thus eliminated, we interpret the action currents as emanating from the only remaining portion of the circuit, the receptor portion. Now both these persisting action currents and the voice waves become greatly reduced in frequency

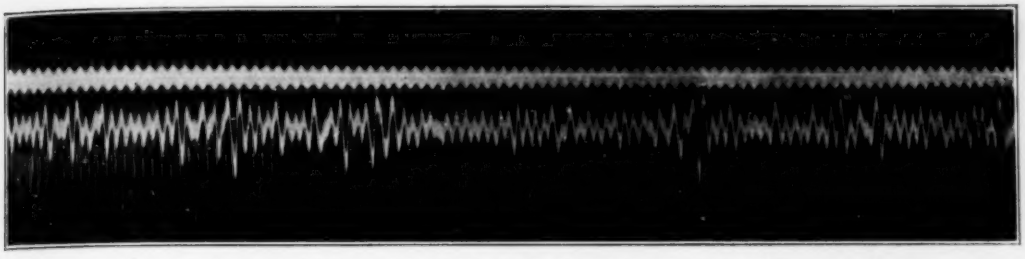

Fig. 1.—Record of the dog's voice and of action currents from the cricothyroid muscle. From above downward are read the time line (0.05 second), the action current line and the voice line.

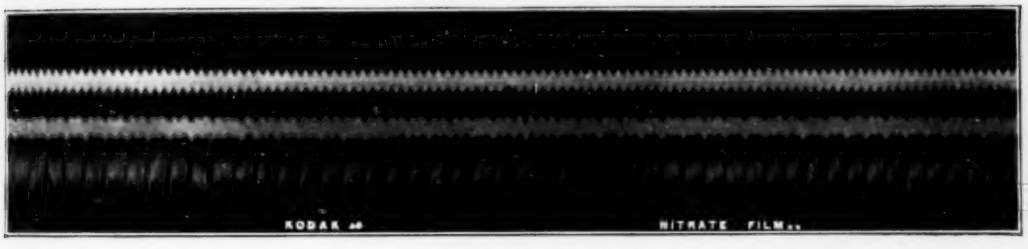

Fig. 2.—Record of the dog's voice and of action currents from the cricothyroid muscle following transection of the nerve. The record is read as in figure 1.

and amplitude. With transection it is to be noted especially that in all records obtained consistent synchronization ruled.

Both before and after the transection there were no action currents to correspond with the majority of the partials in the voice waves. The one exception to this occurred following the transection in which an action current was consistently noted for from every five to seven voice partials.

The technic of this experiment did not lend itself to a comparative study of the intensity of the sound waves and that of the action currents.

When the electrodes were placed in adjacent tissues other than muscle or nerve [4] during phonation, the action currents described did not appear.

Cerebral Observations.—All cortical and subcortical regions employed gave identical results, so that the following observations are true for all of them. The cerebral and the cricothyroid muscle observations are essentially identical. Thus again there is found the striking synchronization between the sound wave frequency in phonation and the cerebral action current frequency. Too, the modification in the former is attended by a corresponding modification in the latter. In phonation the frequency range for the brain is the same as that for the muscle. Finally, the synchronization is not consistently recorded.

When the electrodes were placed in adjacent tissues other than cerebral during phonation, the action currents already described did not appear.

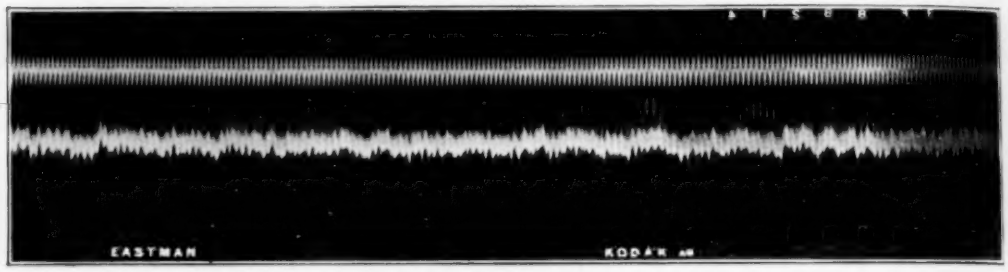

Fig. 3.—Record of the dog's voice and of cerebral action currents. The time is in 0.001 second; otherwise, the record is read as in preceding figures.

To supplement this approach to the understanding of the relation of the stimulus pattern to the mass response, the Galton whistle was substituted for the dog's voice as an intended stimulus. The only difference noted in the results with this stimulation as compared with those obtained with the dog's own voice was the relative infrequency of the synchronizations.

COMMENT

The outstanding ubiquitous coincidence of the sound waves and the action currents furthers an organismic understanding of the responding

4. Lindemann, following our technic in this laboratory, reported observations for the recurrent laryngeal nerve similar to those we herein record for the cricothyroid muscle and the cerebrum (Proc. Soc. Exper. Biol. & Med. **27**:479 [March] 1930). Wever and Bray, working with the auditory nerve of the cat and with the human voice as the stimulus, reported what would seem to be a perfect correspondence between the voice waves and the action currents of the nerve when they transformed the latter back into the identical sound wave stimuli (Science **71**:215 [Feb. 21] 1930).

tissue. In response the cerebral and muscle tissues are seen each as particularlizations of the one responding system. This perspective makes one cautious when considerations not only of the "seats" of cerebral function but also of the "tracts" of the cord are concerned. Rather it leads one to regard the whole reacting system, despite its additive morphology and its earlier assigned "block-built" physiology, as a unit-mass—the total mass being affected by the stimulus although its particular individuations may at one or another time be especially attuned to it. The remarked absence of synchronization likewise noted in all records excepting in those obtained following transection of the nerve to the muscle enriches the observations in an interpretative sense. In the first place it cannot be posited that the activity of the brain and the activity of the cricothyroid muscle prior to the transection were concerned only with phonation. Moreover, the elimination of the concomitant extraneous activity produced by the transection resulted in the recording of our only constant synchronizations. The fact that with the whistle as the intended stimulus the synchronization was relatively infrequent, we take to mean that we were dealing less frequently with an adequate stimulus. It is noteworthy that the whistle stimulus yielded synchronizations in frequencies as high as 2,000 per second.

The stimulus pattern appears to be relayed through the stimulus-response circuit in unaltered form. This fact militates against such concepts as central "smoothing processes," slowing up stations or individual nerve rhythms.

Concerning the genesis of action currents we envisage the energy environing the responding tissue as their source. When this energy impinges on the tissue it is translated by the latter into physiologic form. The most logical locus for this translation we regard as being the so-called receptor portion of the stimulus-response circuit. If this is true, the action currents obtained from muscles are viewed as having been relayed there from the nervous process. Our results demonstrate that the frequencies of these action currents are not altered by such transmission.

In conclusion it remains to test further the concept of unit-mass functioning by attempting to discover similar significant relationships existing between stimulus and response patterns in other nerve tissues not ordinarily envisaged as seriations of given activated stimulus-response circuits.

THE GALVANIC SKIN-REFLEX

FURTHER ASPECTS IN PSYCHOPATHOLOGIC GROUPS *

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In studies of the psychogalvanic reflex and of the electrical skin resistance in groups of persons representing various psychopathologic and normal reaction types, certain definite features have been found to be characteristic of the reaction groups studied. In earlier reports, we compared the average response of these groups to a standard situation, that is, to the reading aloud of a list of stimulus words. It was found that there were relatively few responses in the groups of catatonic, paranoid schizophrenic and depressed persons when compared with the more frequent reactions of normal persons. In the latter, reactions were well connected with the stimulus words, whereas in the psychopathologic cases they occurred more independently and erratically, especially in the paranoid schizophrenic patients, a feature which is well understandable on the basis of the general behavior constellation of these patients.

Richter [1] reported a higher resistance of the skin to electrical currents in catatonic disorders and relatively low resistance in cases of paranoid dementia praecox. Similar results were found by us when the averages of groups of catatonic and paranoid schizophrenic patients were compared. The resistance of normal persons was somewhat higher than that of paranoid schizophrenic patients, while that of depressed patients was still higher, but not as high as the catatonic ones.

The present report is a study of the content of the stimulus words eliciting psychogalvanic responses in persons belonging to these different psychopathologic and normal reaction types. The data were gathered from the experiments that we presented in our earlier papers, though not all of the records of the previous studies are included in the present paper. The method used for these experiments, which were carried on in the Psychological Laboratory of the Henry Phipps Psychiatric

* Submitted for publication, Sept. 9, 1930.

* Read at the Ninth International Congress of Psychology, Yale University, New Haven, Connecticut, Sept. 2, 1929.

1. Richter, Curt P.: The Electrical Skin Resistance, Diurnal and Daily Variations in Psychopathic and in Normal Persons, Arch. Neurol. & Psychiat. **19**:488 (March) 1928.

Clinic, Johns Hopkins Hospital, has already been reported in detail by both Richter [2] and Syz.[3] Since the experimental setting was as consistent as possible, and since we excluded from the calculations on content those experiments in which the stimulus situation showed any marked deviation from the standard, the reaction picture obtained can be considered as a fairly representative behavior response. This behavior response may have been modified in some of the patients by a specific alteration of the physiologic system which is involved in the galvanic skin reaction. The frequent waves, without pertinent provocation and the lack of response to most marked stimuli, which were found in some catatonic cases suggest that in these patients the disturbed functioning of the skin reflex arc may play an important rôle, so that the results in these cases may not represent adequately the response and behavior of the total personality. On the whole, the reaction picture appeared to be pertinently and characteristically related to the experimental setting.

The data were obtained from two groups of normal persons, the one consisting of fifty medical students and the other of twenty-five other normal persons, most of them physicians or professional people connected with the hospital. The data from patients included twenty-five records of depressed patients (eighteen), sixteen of paranoid schizophrenic patients (fourteen) and ten of catatonic schizophrenic persons (seven). The fact that two or three records were obtained from the same person hardly influences the results, as there was an interval of a few months between the first and the subsequent tests.

The stimulus lists (table 1) included 54 words for the students (B) and 107 words for all the other persons (A). In both lists the words were chosen and arranged according to the following principle: Care was taken to include words which would direct the attention to the different fields of human interest, endeavor, concern and sensitiveness, and to so distribute these words throughout the lists that consecutive words were indicative of different topics. In this way we tried to avoid the stimulation of similar emotional and associative complexes by consecutive words and to prevent cumulative effects which would have made the observations less representative and more difficult of interpretation. In the list for students, a few words were used which referred more specifically to the activity and psychologic setting of this particular

2. Richter, Curt P.: Physiological Factors Involved in the Electrical Resistance of the Body, Am. J. Physiol. **88**:596 (May) 1929.

3. Syz, Hans C.: Psychogalvanic Studies on Sixty-Four Medical Students, Brit. J. Psychol. **17**:54, 1926; Observations on the Unreliability of Subjective Reports of Emotional Reactions, ibid. **17**:119, 1926; Psychogalvanic Studies in Schizophrenia, Arch. Neurol. & Psychiat. **16**:747 (Dec.) 1926. Syz, Hans C., and Kinder, E. F.: Electrical Skin Resistance in Normal and in Psychotic Subjects, Arch. Neurol. & Psychiat. **19**:1026 (June) 1928.

group of persons. The name of the subject was read three times during the experiment, near the beginning, midpoint and at the end of the list. The first four (students, list B) or five (list A) words, relatively

TABLE 1.—*List of Words and Frequency of Responses in Percentage of Stimuli Given*

List A—25 Normal Persons

	Per Cent		Per Cent		Per Cent
1. head		37. misspent youth	28	73. poor health	52
2. lake		38. fat body	40	74. coward	32
3. table		39. childish	24	75. pollution	64
4. lazy		40. dishonest transaction	16	76. stupid	44
5. brown		41. frigid	36	77. passionate	60
6. housewife — husband	48	42. prayer	28	78. suicide	44
7. Catholic	60	43. insane	28	79. conceited	44
8. snob	48	44. getting old	16	80. baby	40
9. Negro	24	45. "name"	84	81. friendless	24
10. peach	24	46. extravagance	44	82. sin and damnation	56
11. "name"	88	47. intercourse	64	83. happy home	40
12. successful life	48	48. stingy	28	84. invalid	36
13. lover	64	49. protestant	28	85. birth control	48
14. miserly	44	50. bladder	36	86. cruel	24
15. children	32	51. clumsy	32	87. no future	24
16. hot-headed	68	52. prostitute	56	88. wife	56
17. degenerate	36	53. death	36	89. parasite	32
18. good health	44	54. confession	44	90. divorce	40
19. effeminate	44	55. childhood sex experience	52	91. shabby	20
20. dreams	32	56. conscientious	36	92. happiness	24
21. lonely	44	57. Holy Virgin	40	93. Jesus Christ	40
22. Holy Ghost	48	58. disappointment in love	64	94. wealthy	36
23. financial losses	44	59. cheating	48	95. impotent	40
24. husband	44	60. unforgiven	44	96. attractive	28
25. schemer	8	61. poor sufferer	40	97. disease	28
26. Jewish	36	62. homosexual	60	98. liar	24
27. bad teeth	16	63. deformed	40	99. marital difficulties	36
28. selfish	20	64. piggish	24	100. inferior	20
29. naked body	52	65. pregnant	56	101. menstruation	44
30. bonehead	16	66. bullying	44	102. unpopular	24
31. social failure	40	67. sloppy appearance	24	103. masculine	24
32. self-abuse	56	68. masturbation	60	104. failure in life	20
33. borrowed money	24	69. downhearted	20	105. immoral	24
34. undersized	16	70. Heavenly Father	24	106. old maid	16
35. ambitious	28	71. kiss	68	107. concealed sins	36
36. father	16	72. "name"	72		

List B—50 Students

1. head		19. lonely	18	37. misspent youth	50
2. lake		20. frigid — impotent	26	38. father	22
3. table		21. stubborn	38	39. disappointment in love	52
4. tree		22. sister	32	40. undersized	20
5. housewife — husband	66	23. lazy	28	41. insane	44
6. snob	38	24. "name"	80	42. cheating	28
7. lover	48	25. fraternity	48	43. prostitute	50
8. stingy	42	26. self-abuse	62	44. unpopular	28
9. "name"	84	27. ambitious	22	45. Catholic	40
10. success	44	28. sneak	32	46. "name"	78
11. brother	36	29. mother	28	47. poor health	38
12. kiss	72	30. inferior	28	48. passionate	48
13. the dean's office	62	31. intercourse	68	49. kicked out	44
14. hot-headed	26	32. childish	20	50. weakling	26
15. unpaid bills	36	33. anatomy	36	51. suicide	38
16. naked body	80	34. bonehead	44	52. stupid	18
17. egotism	24	35. unchaste	38	53. Jesus Christ	62
18. Jewish	36	36. clumsy	38	54. immoral	30

neutral in content, were used as a "shock absorber," for familiarizing the subject with the test setting. The reactions to these preliminary words are disregarded in our study of the records, and therefore we have in list A 102 words and in list B 50 words.

After assembling the records we determined the total number of responses for each word in each group of persons and also the average

number of responses per word in each group. It was found that the reactions were distributed throughout the entire list, every word eliciting some responses in each group except the catatonic. The average responses to the words calculated as percentage of the total possible responses to each word is given in table 1 for the groups of students and for the other normal persons.

The average frequency of responses for the entire list was also determined, and was found to vary from group to group, indicating that the general responsiveness to the stimulus situation differs in the different groups of subjects. This general reactivity to the entire list of words was again calculated as percentage, that is, reactions obtained compared with stimuli given. The figures for the various groups were as follows: student, 41.9; normal, 38.6; depressive, 23.3; paranoid schizophrenic, 21.3, and catatonic, 6.8 per cent (table 3). It is of interest to note that the two groups of normal persons agreed closely in their general reactivity, though the stimuli presented to them were different. A slightly higher reactivity in the case of the students may have its explanation in the fact that the average age of the students was lower than that of the other normal subjects and that the students were probably more sensitized to the test setting which for them had somewhat the character of an examination setting. The slightly lower general responsiveness of the twenty-five normal persons is apparently not due to their longer list of stimulus words and to a lowering of reactivity toward the end of the list, as the number of reactions to the first fifty-one words was practically the same as that of the reactions to the second half. The slight divergence of the average frequency of responses in the different groups from that given in earlier publications results from the fact that the data of the present study deal only with part of the records used in these other studies.

Not only in the average reactivity is there a striking agreement in the two groups of normal persons but also in the type of words which elicited especially frequent responses. As may be seen in table 1, some of the particularly effective stimulus words (name, sex words) had almost the identical percentage of responses in the two normal groups. This is the more interesting as this agreement stands out in sharp contrast to the results in the psychopathologic patients.

For further comparison, the words were grouped according to the following topics: the name, words suggestive of sex activities or relationships, words which might be expected to arouse the consciousness or concern about one's own self, words of a depressive content, words relating to success and words of a religious nature (table 2). Such a classification is of course somewhat arbitrary, and a certain overlapping is inevitable. A few words did not fit into any of these groups, and therefore they are not considered further in our calculations.

It was found that the different groups of persons differ from each other in the frequency of response to the different classes of content. While this is true if the average reactivity to each class of content is computed, it is still more outstanding if only the words are considered

TABLE 2.—*Words Classified According to Content*

	List A		List B
Name	name		name
	name		name
	name		name
Sex	peach	pollution	wife — husband
	lover	passionate	lover
	effeminate	baby	kiss
	naked body	birth control	naked body
	self-abuse	husband	frigid — impotent
	frigid	wife	self-abuse
	intercourse	divorce	intercourse
	prostitute	impotent	unchaste
	childhood sex experience	marital difficulties	disappointment in love
	disappointment in love	menstruation	prostitute
	homosexual	masculine	passionate
	pregnant	immoral	immoral
	masturbation	old maid	
	kiss	concealed sins	
Self	snob	stupid	snob
	miserly	conceited	stingy
	hot-headed	parasite	hot-headed
	schemer	cruel	egotism
	selfish	shabby	stubborn
	bonehead	attractive	lazy
	childish	liar	sneak
	extravagant	bad teeth	inferior
	clumsy	fat body	childish
	conscientious	undersized	bonehead
	cheating	getting old	clumsy
	deformed	stingy	undersized
	sloppy appearance	piggish	cheating
	coward	bullying	unpopular
			weakling
			stupid
Success	successful life	happiness	success
	good health	wealthy	dean's office
	ambitious	financial losses	unpaid bills
	happy home	borrowed money	ambitious
			kicked out
Religious	Catholic	confession	Jewish
	Holy Ghost	Holy Virgin	Catholic
	Jewish	Heavenly Father	Jesus Christ
	prayer	sin and damnation	
	protestant	Jesus Christ	

which evoked an especially high number of responses. Tables 3 and 4 give summaries of these observations.

As the general reactivity varies for the different groups of persons, it would not be adequate to compare directly the averages of responses (percentage) which a word has elicited in the different groups. As a matter of information, we give this average response in percentage in table 3 with the words listed in this table. In addition, however, it appeared necessary to relate these averages to the general reactivity.

TABLE 3.—*Words Eliciting Especially Frequent Responses, At Least One Third More than the Average for the Group (One Third Plus Reactivity)* *

	Student (Short List)	Normal	Depressive	Paranoid	Catatonic
General reactivity	41.8%	38.6%	23.4%	21.3%	6.8%
Name	84 name	88 name	64 name	55 name	
	80 name	84 name	60 name	54 name	
	78 name	72 name	52 name		
Sex	80 naked body	68 kiss	44 lover	59 naked body	pregnant
	72 kiss	64 lover	36 self-abuse	44 childhood sex experience	baby
	68 intercourse	64 intercourse	36 masturbation		effeminate
	66 wife	64 disappointment in love	32 divorce	31 intercourse	
	62 self-abuse			31 kiss	
		64 pollution		31 birth control	
		60 homosexual			
		60 masturbation			
		60 passionate			
		56 self-abuse			
		56 prostitute			
		56 pregnant			
		56 wife			
		52 naked body			
		52 childhood sex experience			
Depressive	.....	52 poor health	52 insane	56 downhearted	death
			48 death	38 insane	
			44 suicide	38 lonely	
			40 degenerate		
Self	.....	68 hot-headed	36 deformed	38 bonehead	selfish
		48 snob	36 cruel	38 clumsy	cruel
		46 cheating	32 miserly	38 coward	bullying
			32 clumsy	31 selfish	
			32 coward	31 extravagant	
			32 dishonest transaction	31 fat body	
Success	62 dean's office	48 successful life	44 successful life	38 ambitious	successful life
			32 financial loss	38 financial loss	good health
				31 successful life	borrowed money
				31 happy home	
Religious	62 Jesus Christ	60 Catholic	44 Catholic	31 Catholic	Catholic
		56 sin and damnation	36 sin and damnation		
		48 Holy Ghost	32 Holy Ghost		
			32 prayer		
			32 Heavenly Father		

* Words with at least two-thirds plus are underlined, words with plus reactivity of 100 per cent or more are underlined twice. The numbers indicate the average response to the individual words as percentage of responses possible.

That is, the reactivity of each group of persons to the entire list of stimulus words was taken as a basis for determining which of the words elicited in each group a higher number of responses than would be expected. We include in table 3 all those words which had a plus percentage of at least one third more than the general reactivity. For instance, for the word "passionate" in the normal group: the general reactivity of the normal subjects was 38.6 per cent, their reactivity to the word "passionate" was 60 per cent, which gives a plus percentage of 21.4, or more than one third of 38.6. Those words in which the frequency of response was at least two-thirds greater than the average for all words are underlined in the list, and those in which the frequency showed a plus percentage of 100 or more, that is, those which elicited at least twice the average reactivity are underlined twice. In the catatonic group, a slightly different tabulation seemed advisable on account of the general rarity of reactions. Even one response per word

TABLE 4.—*Percentage of Words in Each Class Eliciting at Least One Third More Than Average Number of Reactions* *

	Student	Normal	Depressive	Paranoid	Catatonic
Name	100	100	100	67	0
Sex	42	50	14	18	11
Depressive	0	5	21	16	5
Self	0	10	20	20	11
Success	20	13	25	50	38
Religious	33	30	50	10	10

* This table indicates the relationship between tables 2 and 3.

is considerably more than the average (6.8 per cent) for the ten records, and therefore we included in the table for this group only those words which elicited two or three responses; the underlining also is omitted in these records.

In table 3 it may be seen that there are marked differences in the type of content which was especially emphasized by the various groups. In general, it is evident that the two normal groups are very similar in the responses to different topics, in spite of the different stimulus lists. In both there is a definite emphasis on words relating to sex and a low frequency of response to words indicating depressive topics, concern about self and success. In the psychopathologic groups, on the other hand, there is less emphasis on sex terms but a marked reaction to words that are apt to arouse depressive and self-conscious preoccupations, the greatest emphasis on depressive contents being evident in the group of depressed persons. But on the whole, the difference in this respect between the depressive group and the paranoid schizophrenic group was not marked. Only in regard to religious topics was there a much larger response among the depressed patients. The name elicited a high

number of responses in the two normal groups, and the lower response in depressive and paranoid groups is about proportionate to the generally lower reactivity in these pathologic groups; disproportionately low in this respect was the catatonic group, in which only one response to the total of thirty name stimuli which were presented to these patients was noted. The various classes of words as listed in table 2 differ markedly in the number of words included, from three (names) to twenty-eight (self, list A). Therefore, it seemed to be of interest to know also the percentage of words in each class that gave a high reactivity. Table 4 shows this percentage; that is, the words of more than one-third plus reactivity compared with the total number of words in each class. The results listed in table 4 corroborate the results shown in table 3; the reported emphasis in regard to different contents is evident not only if the most important stimulus words are considered, but also if the less effective words are included in the calculation.

COMMENT

From the vast amount of experimental work done with the psychogalvanic reflex, it has been concluded that in general this reaction of the skin is indicative of a heightened emotional tone, though according to some investigators other psychic operations (sensory impressions, intellectual and conative processes) and physiologic activities (breathing, vasomotor functions, local changes in permeability in the skin) also may elicit or influence this response; it has also been reported that there may be emotional reactions that are not accompanied by this reflex. But in the main, the evidence generally established corroborates the conception that the galvanic responses to the word stimuli are part of a temporarily increased emotional tension, a state that is, of course, closely intermingled with and dependent on action tendencies aroused by the word and its associations.

In the present study we did not make a careful examination and analysis of each case. But in a number of persons, especially of the normal groups, a short analysis of the various responses was made. It was found that in many instances the emotional reactions to the word stimuli were clearly recalled; it was reported for instance that the stimulus word led to the recollection of specific occurrences, the emotional tone of which was also partly reexperienced. In other instances, it was evident that it was more the relation of the ideational content to the experimental setting which was of importance and caused the affect swing, which then appeared to be in the nature of surprise, resentment and embarrassment. The sudden and often startling focusing of attention in itself embodies an emotional feature which was especially evident in the reaction to the calling of one's name. On still other occasions

the subjects were not clearly aware of any definite emotional response, but from an analysis of the specific contents as they related to the individual and to the test setting, it was evident in most cases that this content was not without a special meaning and importance, that it was linked up with specific though often disguised and buried strivings, sensitivities, emotional conflicts, "complexes." Conflicting counterimpulses may not only set up an affect state, but they are also active in the general tendency to repress or evade undesirable aspects of experience and to disperse and eliminate immediately the affect reactions which might be activated by these specifically sensitized contents. This trend, with its evasion, disguise and self-deception, naturally embodies also an impediment to a correct and conscious evaluation of one's own emotional experiences. But this repressive factor may be realized by the examiner, and if its influence is reckoned with in the interpretation of the observations, it becomes still more evident how closely the galvanic reactions of the skin are linked up in our experiments with emotionally colored processes.

While the psychogalvanic reactions may be taken as indicative of an emotional stirring, the specific type of this emphasis is not revealed by the reflex. It is not evident in what way a stimulus word is important for the subject. The emotion may represent the affect contained in the activated complex itself, or it may be elicited rather by the relation of the associative trends to the immediate test setting. In either case the response shows that the topic indicated by the stimulus word is of some importance to the individual, that it is linked up with his wishes and desires, no matter whether the momentary constellation points in the direction of fulfilment or contains rather a conflict, the result of counterimpulses which may be deeply engrained in the patient's emotional constellation or which may be more superficially aroused by suggestions embodied in or projected into the experimental situation.

In the psychopathologic groups there is outstanding emphasis on words relating to personal characteristics, to terms which imply a valuation of one's self, often with an undertone of expected failure, inferiority or guilt. The results as listed in table 3 show clearly that these egocentric and anxious preoccupations, which characterize the neurotic or psychotic state, were brought out objectively by our experiments. Though the number of records in the psychopathologic groups was relatively small, their averages differed so markedly from those of normal persons that we feel justified in considering the general trend as rather significant. From our present limited material, it could not be determined conclusively in how far one might differentiate various types of disorder, for example, an outspoken depressive state from conditions of psychobiologic disorganization that do not present such sweeping and

clearcut emotional trends. There was a slightly greater emphasis on depressive topics among the depressed patients, but for a more definite determining of such finer differentiations, it would be necessary to extend these experiments and statistics over larger groups of well chosen psychopathologic cases.

Considered quantitively, it is noteworthy that the paranoid schizophrenic patients showed almost the same general reactivity as the depressed ones. As the psychogalvanic response may be taken as an indicator of emotional activity, it appears in fact that there is more emotional activity in the schizophrenic than in the depressive group, as the frequent spontaneous and disconnected galvanic waves in the paranoid schizophrenic group were not included in computing the general reactivity (21.3 per cent). One may see in these experimental results a confirmation of the view expressed by various investigators who proceeded more from clinical and analytical data, namely, that the schizophrenic development often does not mean primarily a lack and decrease of emotional assets and activities, but rather a disturbance in the constellation and application of emotional trends.

As already mentioned, our experiments show that in the normal persons there is less emphasis on egocentric anxieties and concerns. There is, however, a marked predominance of reactions to sex words. As might be expected, and as was evident from our discussions with our subjects, this is due largely to a socially induced sensitiveness. The social situation with its self-consciousness and prohibition in regard to sex matters is reduplicated by the experimental setting, and accounts in a large degree for the special emphasis of this topic.

Thus, if the results from groups of normal and psychopathologic subjects are compared, we find that the psychogalvanic reflex shows somewhat specific differences, not only in the frequency and type of waves, but also in the content to which reactions were elicited. The topics emphasized suggest an emotional preoccupation which is in accord with the behavior pictures of these different personality groups.

MONOBROMATED CAMPHOR

A STANDARDIZED CONVULSANT *

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The experimental production of fits by drugs has been a particularly interesting branch of research in epilepsy. Many workers, especially Magnan,[1] Horsley, Boyce, Russell, Türck, Turtschaninow, Purkinje and Muskens [2] have used various drugs and attempted to explain epileptiform phenomena on the basis of the effects produced by these drugs. However, these researches have failed to reveal any one drug that will cause effects exactly comparable with genuine epilepsy. Of all the drugs used, camphor or its derivatives appears to be the substance best suited for the experimental study of fits.

For many years, camphor has been known to produce convulsions in warm-blooded animals, and not infrequently in man through an overdose. Alexander, in 1829, and later Wiedemann [3] (1877) described the convulsive effects of the drug as "epileptiform." Later, Schwartz (1871), Gubler (1874), Pathault [4] (1876) and Lawson [5] (1897), described its effects on the nervous systems of man and of animals. More recently, Muskens has made use of camphor in his excellent studies on epilepsy. The results obtained with it experimentally are encouraging, because the fits produced by its use are of the myoclonic type, and are similar to genuine epilepsy.

CHEMICAL DATA

Camphor, $C_{10}H_{16}O$, is a solid derivative of terpene, $C_{10}H_{16}$, which is allied to the volatile oils. It is prepared by the distillation of the wood of the camphor tree, and this form of natural camphor is dextrorotatory. A synthetic camphor has also been prepared which is racemic,

* Submitted for publication, Nov. 3, 1930.

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1. Magnan, V.: Recherches sur les centres nerveux, Paris, G. Masson, 1876.
2. Muskens, L. J. J.: Epilepsy, New York, William Wood & Company, 1928.
3. Wiedemann, C.: Arch. f. exper. Path. u. Pharmakol., 1877, vol. 6.
4. Pathault, L.: Du bromure de camphre (camphre monobromé de Wurtz) et de ses usages thérapeutiques, ed. 2, Paris, A. Delahaye, 1876.
5. Lawson: Brit. M. J. **2**:660, 1897.

and a levorotatory type occurs. The "levo" form is said to be somewhat more active on the nervous system. In large doses, camphor produces convulsions in warm-blooded animals.

Volatile or essential oils have also been used to produce experimental convulsions. These oils consist of the odorous principles of vegetable organisms and contain certain terpenes, camphors and related compounds. Among the more important are the oil of wormwood, the oil of fennell, and the oil of absinth. The active principles contained in the oil of fennell and the oil of absinth are fenchone, $C_{10}H_{16}O$, and thujone, $C_{10}H_{16}O$, respectively; the latter are both isomers of camphor.

Other drugs, such as strychnine and picrotoxin, have been used to produce convulsions in animals, but are not germane to this study.

The exact mechanism of the convulsant action of camphor and the volatile oils has been disputed: some believe that the reaction, like that of a neurotoxin, is due to cerebral cellular irritation; others believe that the convulsions are due to the effects on the cerebral vasomotor system, causing constriction of the cerebral vessels and secondary cortical anemia. D'Ormea[6] (1903), investigating effects of the volatile oils, believed that their intravenous injection caused a dilatation of the cerebral vessels. McDonald and Cobb, studying the effects of absinth, observed cortical vasoconstriction just preceding a convulsion. Investigations by Rimini (1900), Hildebrandt (1901), Matzel (1905) and Geinitz (1912) have shown that stimulation varies with the different oils; e. g., turpentine has scarcely any effect, while absinth, fenchone, thujone, carvon and camphor produce violent epileptiform convulsions. Depression of the central nervous system has been more uniformly observed; e. g., eucalyptus, mint, rosemary, citrol and valerian diminish reflex excitability so that large doses are able to prevent convulsions produced in rabbits by strychnine. The proportionate content of the ingredients of the essential oils varies in different samples and thereby has led to some confusion in experimental data from different laboratories.

STANDARD SOLUTION AND TECHNICAL PROCEDURE

It has therefore seemed necessary to standardize a substance of uniform quality, capable of producing fits in animals, that could be used with uniform results in different laboratories. We have elected to standardize a solution of monobromated camphor for its convulsant properties. Monobromated camphor is $C_{10}H_{15}OBr$ (=231.2). The effects produced are identical with those of pure camphor. The standard solution adopted consists of 10 Gm. of monobromated camphor (U. S. P) dissolved in 100 cc. of 95 per cent alcohol. We have used the cat as the experimental animal.

6. d'Ormea, A.: Arch. ital. de biol. **40**:141, 1903.

The cat was placed on its back on the operating table, and kept in place by ties holding each limb loosely. Towel bands were placed about the neck and body of the animal. A solution of 1 per cent procaine was injected into Scarpa's triangle of one thigh; the femoral vein was exposed and the internal saphenous nerve was cut so that the skin area was desensitized during the experiment. A tuberculin syringe, graduated in one-hundredths of a cubic centimeter, and a fine hypodermic needle were used for injecting the standard solution of monobromated camphor. A few cubic centimeters of physiologic solution of sodium chloride was injected directly following the administration of the solution of camphor to insure all the material getting into the general circulation.

DESCRIPTION OF THE FIT CAUSED BY MONOBROMATED CAMPHOR

In the epileptic fit produced by the intravenous injection of monobromated camphor the following reactions are easily observed in their order of occurrence:

1. Immediately following the injection of a sufficient quantity of the drug, the respiratory rate is increased. With very large doses respiration may cease for from five to fifteen seconds. On one or two occasions the animal suddenly died.

2. A prodromal period ensues in which small, spontaneous convulsions or shocks occur with extreme rapidity and ease.

3. There is a period of labile excitability. (In this period, as Muskens suggested, important changes probably take place in the function of conduction. Two of us have observed slight cortical blanching in this stage.)

4. This stage is followed by a clonic or tonic fit, depending on the dosage, the larger doses giving more of the tonic type of fit. The fit usually begins with a series of very rapid and severe convulsions which individually are probably clonic. There are powerful and rapidly successive muscular contractions in this stage.

5. In this stage isolated clonic seizures are discernible.

6. Respiration ceases during the tonic stage. After the clonic fit a regular respiratory rhythm is resumed.

7. There is usually frothing at the mouth.

8. After the severe clonic convulsive seizures cease, automatic movements occur, e. g., jumping or movements of progression, followed by signs of fear or excitement in the cat.

9. During this stage there is a diminution or, more often, a loss of reflex excitability, wherein the corneal, the pinnal and the deep reflexes are not elicitable.

10. Involuntary evacuation of the bladder and bowel may occur in any stage, usually in the stages 2 to 6.

11. In male cats, erection of the penis is often seen.

During the convulsions the pupils dilate and do not respond to light; the hair of the back is erect and the tail is bushy; the pads of the feet glisten with moisture, and the mucous membranes of the mouth and nose are blanched just preceding the fit. There is usually a cry just before the onset of the convulsive seizure.

COMPARISON WITH EPILEPTIC FITS IN MAN

The essential similar characteristics of a true epileptic fit occurring in patients suffering with epilepsy and in animals (cats) under suitable dosage of monobromated camphor are:

1. Rapid disordered convulsive discharges occur in different parts of the body. These are seen especially in myoclonic types of epilepsy.

2. There are attacks of petit mal, associated with periods of temporary confusion, occasionally associated with enuresis and some dilatation of the pupils. These minor attacks are difficult to determine in animals; but a cat can often be observed to stare about, and to have a fleeting pupillary dilatation and coincident enuresis.

3. There are attacks of grand mal, associated with loss of consciousness, pupillary dilatation, convulsive seizures and loss of sphincteral control, all of which are seen in the cat under adequate doses of monobromated camphor.

4. A loss of reflex excitability is experienced in the period directly following a major seizure. The corneal reflex and the deep reflexes are not elicitable in this stage. This is a well established clinical fact, and is seen in the cat in the corresponding "postepileptic state."

5. Frothing at the mouth, respiratory alterations, sweating of the body, and also of the foot pads in animals, and, occasionally, biting of the tongue are seen.

6. There appears to be a tendency (as observed by Muskens and also seen in this laboratory) for both man, when suffering with epilepsy, and the cat, when given monobromated camphor in oil by stomach, to develop fits at the periods of awaking or of falling asleep. This may be related to associated variations of the intracranial pressure at these times.

7. After the major fit, the pupils return to normal size, respiration becomes regular in rhythm, and the animal becomes, after a short while, more alert to his surroundings.

It is apparent from these phenomena that furthur studies of fits produced by monobromated camphor are of value because of their similarity to genuine epilepsy seen in man.

STANDARDIZATION OF SOLUTION

The standard solution used contains 10 Gm. of monobromated camphor (U. S. P.) dissolved in 100 cc. of 95 per cent ethyl alcohol. Muskens used this drug dissolved in oil and established a rather rough dosage of from 0.5 to 1 Gm. of monobromated camphor by stomach, irrespective of the weight of the animal. The problem of adequate absorption by this route is obvious. He found that 0.5 Gm. by mouth produced a tendency to shocks, and larger doses produced severe fits or death.

Standardization of Monobromated Camphor

Experiment	Cat*	Weight, Pounds	Weight, Kg.	Twitches Obtained, Total Solution, Cc.	Minimal Convulsant Dose, Total Solution, Cc.	Lethal Dose, Total Solution, Cc.	Clonic Convulsions	Tonic Convulsions	Total Time, Minutes	Total Solution Used, Cc.	Minimal Convulsant Dose, Cc. per Lb. of Cat	Minimal Convulsant Dose, Cc. per Kg. of Cat	Minimal Lethal Dose, Cc. per Lb. of Cat	Minimal Lethal Dose, Cc. per Kg. of Cat	Number of Injections
1	N. C.	9.0	4.09	0.20	0.25	0.40	4	1, 1†	48	1.50	0.028	0.061	0.040	0.097	5
2	O. C.	6.0	2.72	0.12	0.15	0.25	9	1†	102	1.10	0.025	0.059	0.041	0.092	3
3	N. C.	6.0	2.72	0.11	0.15	0.24	5	1†	78	1.25	0.025	0.055	0.040	0.088	7
4	N. C.	8.0	3.63	0.16	0.20	0.24	1	2, 1†	25	0.60	0.025	0.055	0.030	0.066	3
5	O. C.	7.0	3.18	0.14	0.14	0.21	6	1, 1†	76	0.53	0.020	0.044	0.030	0.066	3
6	N. C.	7.5	3.41	0.11	0.11	0.30	7	1, 1†	69	1.33	0.015	0.033	0.040	0.088	7
7	N. C.	5.0	2.27	0.08	0.09	0.22	7	2, 1†	95	1.32	0.018	0.040	0.044	0.096	9
8	N. C.	8.0	3.63	0.18	0.20	0.32	8	1†	39	1.51	0.025	0.055	0.040	0.088	7
9	N. C.	8.25	3.75	0.15	0.17	0.37	6	1†	86	2.25	0.021	0.045	0.044	0.097	9
10	N. C.	10.0	4.54	0.15	0.18	0.45	3	1†	51	1.40	0.018	0.040	0.045	0.099	5
11	N. C.	5.5	2.5	0.10	0.10	0.25	5	1†	65	0.74	0.018	0.040	0.045	0.099	4
12	N. C.	9.5	4.32	0.14	0.19	0.38	5	1†	72	1.57	0.020	0.044	0.040	0.088	6
13	N. C.	7.25	3.27	0.145	0.18	0.26	4	0	55	1.08	0.025	0.055	0.035	0.0.9	6
14	N. C.	8.0	3.63	....	....	0.40	5	1, 1†	54	0.76	.....	.....	0.050	0.110	3
15	N. C.	7.0	3.18	....	....	0.49	8	1, 1†	111	2.69	.....	.....	0.070	0.154	7
16	N. C.	5.0	2.27	....	....	0.28	0	1†	.8	0.28	.....	.....	0.055	0.123	1
17	N. C.	7.0	3.18	....	....	0.42	1	0	21	0.42	.....	.....	0.060	0.132	1
18	N. C.	9.5	4.32	....	....	0.665	1	1†	5	0.665	.....	.....	0.070	0.154	1
19	N. C.	7.0	3.18	....	....	0.56	0	1†	10	0.56	.....	.....	0.080	0.176	1
20	N. C.	6.5	2.95	....	....	0.55	2	1†	20	0.55	.....	.....	0.085	0.187	1

* N. C. indicates normal cat; O. C., operated cat (left parieto-occipital lobe removed).
† Tonic extension preceding death.

We have determined the minimal convulsant dose of monobromated camphor required per pound (or per kilogram) of cat. This standardization we believe is of value in further studies of convulsions. We are at this time completing a study of the effects of other drugs in augmenting or diminishing fits caused by the administration of monobromated camphor.

We have used the intravenous route because of its certainty and have thus eliminated problems of absorption. One one-hundredth (1/100) cubic centimeter of the standard solution contains 1 mg. of monobromated camphor. The following were observed:

1. The minimal convulsant dose per pound of adult cat averaged 0.022 cc. of the standard solution (variations of from 0.015 to 0.028 cc.) with single dose injections.

2. The minimal convulsant dose per kilogram of cat averaged 0.048 cc. of the standard solution (variations of from 0.033 to 0.061 cc.) with single dose injections.

3. The minimal lethal dose per pound of cat averaged 0.067 cc. of the standard solution (variations of from 0.050 to 0.085 cc.) with single dose injections.

4. The minimal lethal dose per kilogram of cat averaged 0.148 cc. of the standard solution (variations of from 0.110 to 0.187 cc.) with single dose injections.

5. With repeated injections of the standard solution at intervals of ten minutes, increasing 0.5 mg. per pound of cat per dose, the lethal dose per pound of cat averaged 0.0395 cc. of the standard solution.

6. Single myoclonic twitches were obtained with doses just below the minimal convulsant dose per pound of animal (see table).

7. The animals died of respiratory failure, the heart continuing to beat for several minutes after respiration ceased.

8. With small doses, one fit was produced. When the dose of monobromated camphor per pound of animal was increased, a series of seizures ensued and in some cases the animal died in status epilepticus.

9. A single large dose caused death (due to sudden cessation of respiration) in more instances than did the total amount of monobromated camphor injected intravenously over a period of time.

10. It required from twenty-four to forty-eight hours for the animal to be completely rid of the effects of the injection of monobromated camphor.

11. After injection of large doses, when the animals had had several convulsive seizures, they appeared to have transient blindness (probably cortical, because the pupillary reactions to light were retained). They walked into objects placed in their path and walked along a wall when it was near. This phenomenon passed off in about ten minutes.

12. Pregnancy or disease influenced the minimal convulsant dosage.

SUMMARY

A solution of monobromated camphor has been standardized for its convulsant properties. The minimal convulsant and minimal lethal doses per pound (or per kilogram) of cat have been accurately established.

Epilepsy in man and convulsions produced by monobromated camphor in the cat are compared.

KNEE JERK

IX. THE EFFECTS OF STIMULATION OF SOME ABDOMINAL VISCERA ON THE PATELLAR JERK *

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AND
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The literature contains numerous accounts of the effects on the thoracic and abdominal viscera of stimulation of the exteroceptors. There are, however, few accounts of the effects on the neuromuscular apparatus of stimulation of the interoceptors. The early meager literature is referred to in a study of skeletal reflexes induced by stimulation of the visceral afferent nerves in the frog and the turtle.[1] In a previous paper of this series [2] evidence was presented in support of the view that mechanical or electrical stimulation of the pulmonary fibers of the vagus nerve might exert an inhibitory effect on the nerve center controlling the knee jerk.

The present paper purports to show that stimulation of the afferent fibers coming from the abdominal viscera may modify the knee jerk either by acting on the nerve center itself or by causing such changes in the skeletal musculature that the patellar jerk is either markedly reinforced or completely prevented.

METHODS

Dogs under various stages of barbital sodium anesthesia were used. In some the spinal cord was left intact; in others the cord was sectioned in the lower thoracic region.

The animals were arranged so that simultaneous records of the knee jerks and the carotid arterial blood pressure could be taken. The knee jerks were studied by means of a special apparatus previously described.[3] It will be recalled that by means of this device it is possible to deliver a constant blow to the patellar tendon every seven seconds over a long period of time, and to record the responses produced.

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* Submitted for publication, Nov. 11, 1930.

* From the Hull Physiological Laboratories of the University of Chicago.

1. Carlson, A. J., and Luckhardt, A. B.: Studies on Visceral Sensory Nervous System; Skeletal Reflexes Induced by Stimulation of Visceral Afferent Nerves in Frog and Turtle, Am. J. Physiol. **55**:366 (April) 1921.

2. Johnson, C. A., and Luckhardt, A. B.: Studies on Knee Jerk; Effect of Raised Intrapulmonary Pressure upon Knee Jerk, Blood Pressure and State of Consciousness, Am. J. Physiol. **83**:642 (Jan.) 1928.

3. Johnson, C. A.: Studies on Knee Jerk; Simple, Dependable and Portable Knee Jerk Apparatus for Use on Higher Mammals and Man, Am. J. Physiol. **82**: 75 (Sept.) 1927.

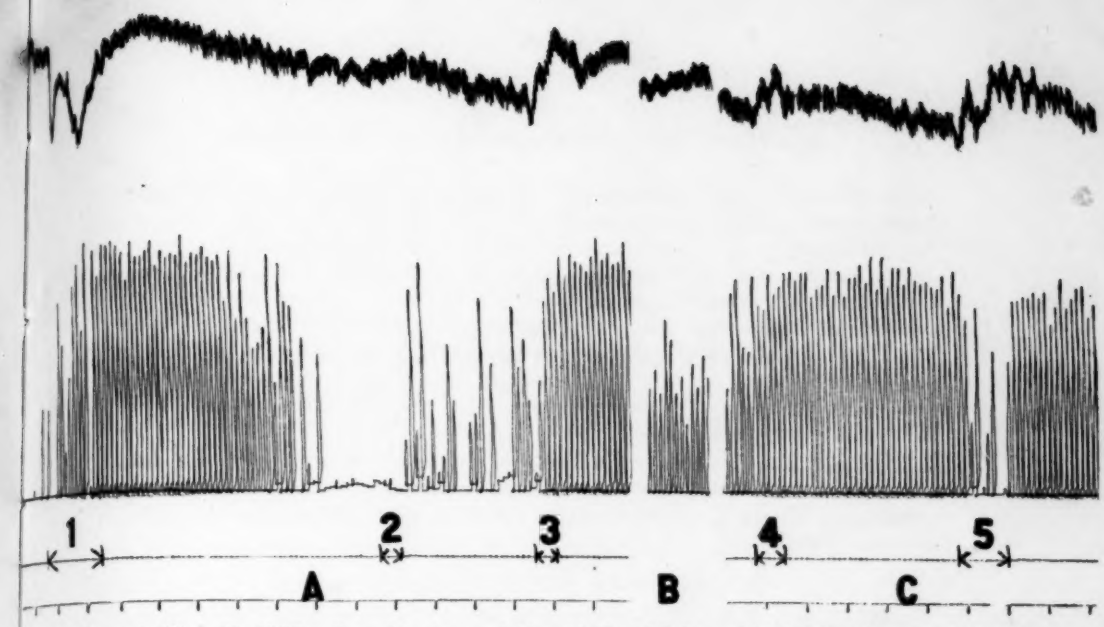

Chart 1 (Nov. 23, 1927).—Into this dog, weighing 10 Kg., 2.5 Gm. of barbital sodium was injected intraperitoneally. Reflexes were produced as shown in the curve: *(A) 1,* by opening the abdominal cavity; *2,* by rubbing the skin around the abdominal wound, which produced shivering and clonic movements; *3,* by light prodding in the abdominal cavity with an ether thermometer, which caused the shivering and clonic movements to stop. The curve shown in (*B*) was produced five minutes after that in (*A*); the animal was shivering and the clonic movements were present. The curve in (*C*) was produced four minutes after that in (*B*) *4,* insertion of the hand into the abdominal cavity and *5,* prodding with a thermometer.

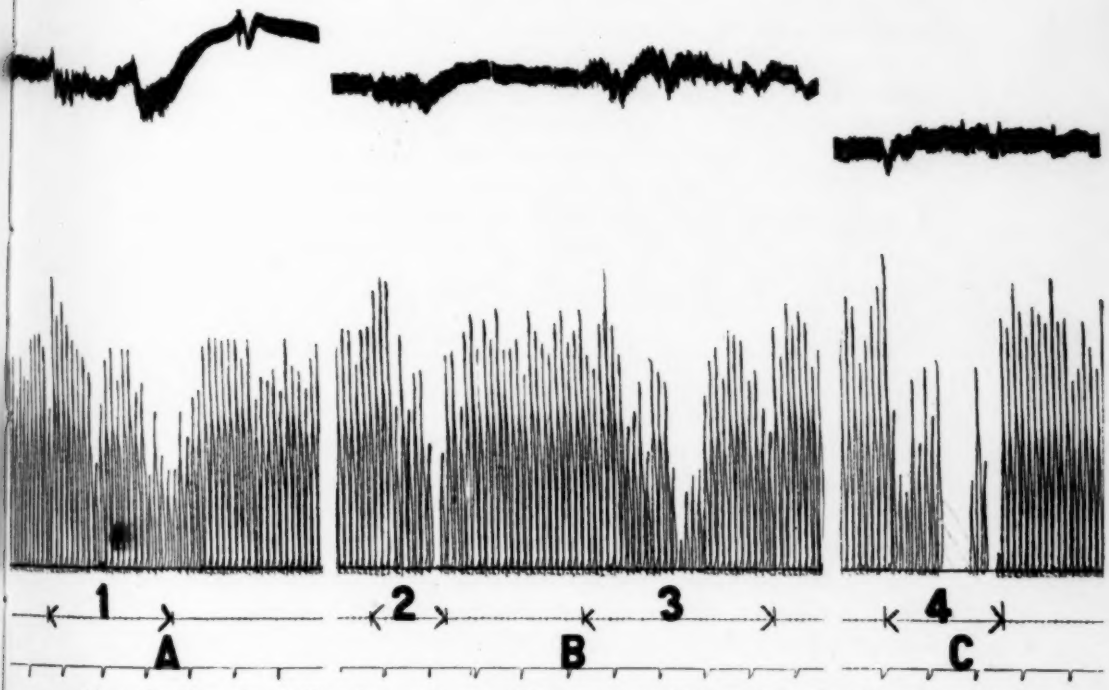

Chart *2* (Nov. 31, 1927).—Into this dog, 275 mg. of barbital sodium per killogram of body weight was injected. Reflexes were produced as shown in the curves: (*A*) *1,* by opening the abdomen; (*B*) *2,* by manipulation in the abdominal cavity (produced twenty minutes after *A*); *3,* by manipulation in the abdominal cavity; (*C*) *4,* by traction on the colon at the proper instant (one and one-half hours after *B*).

After suitable control periods, i. e., a period in which from 200 to 300 responses were recorded (from one half to three quarters of an hour), the abdomen was opened rapidly by an incision extending from the xiphoid cartilage to the symphysis pubis, and the effect on the blood pressure and the knee jerk was noted. Various methods of mechanical stimulation were next applied to different portions of the bowel and some other abdominal viscera. During these manipulations of the abdominal viscera, we also observed the hamstring and quadriceps muscles for visible evidence of contraction. The bowels were pinched or rubbed. Traction was applied to the root of the mesentery of the small intestines and particularly to the mesentery of the rectum.

To check our interpretation of the results obtained, the sciatic nerves of both legs were isolated and sectioned near their exit at the sciatic notch. After suitable control periods, we stimulated the peripheral and central ends of these nerves and recorded the effects.

RESULTS

When the abdominal cavity was opened, we obtained either augmentation (chart 1, *A*, 1) of the knee jerk (seen chiefly in lightly anesthetized animals) or depression (chart 2, *A*, 1) of it (seen chiefly in deeply anesthetized animals).

In a lightly anesthetized animal, slight prodding of the abdominal viscera with a blunt instrument (chart 1, *A*, 3 and chart 1, *C*, 4) caused an augmentation of the knee jerk, while in more deeply anesthetized animals it had little or no effect. More vigorous manipulation of the viscera, especially of the large bowel, the urinary bladder and the gallbladder, usually caused inhibition of the jerk (chart 1, *C*, 5 and chart 2, *B*, 2 and 3). In one animal the large bowel was so sensitive that the lightest rubbing of its surface with a probe or a finger moistened with physiologic solution of sodium chloride was sufficient to abolish the knee jerk completely. It was also found that inhibition of the knee jerk could be obtained more readily by applying traction on the mesentery a fraction of a second before the hammer struck the patellar tendon (chart 2, *C*, 4). It is important that the traction be applied at just the proper instant before patellar stimulation to obtain the maximum effect.

The hamstrings were observed at the time of sudden traction on the viscus, and often they were seen to contract. The sciatic nerve was sectioned near its exit from the sciatic foramen on the leg from which the knee jerk was being taken in order to prevent contraction of the hamstring muscle. Following this, traction or manipulation of the viscera sometimes produced inhibition, sometimes augmentation. At other times no effect was observed.

Stimulation of either the central or the peripheral ends of the sciatic nerve with a faradic current caused complete inhibition. In the former instance, the flexion reflex leads to a diminution of tone of the

antagonistic extensors (quadriceps), which may account for the inhibition; in the latter case the marked flexion mechanically prevents extension of the leg at the knee.

COMMENT

For an adequate explanation of the results obtained in this study it is necessary to consider various factors that modify the amplitude of the knee jerk obtained as a result of a blow to the patellar tendon. The more important factors are: (1) mechanical, and (2) reflex effects acting on the nerve center of the knee jerk.

From a purely physical point of view, the position of the limb is extremely important. There is an optimum position of the leg at which a given submaximal blow will produce a maximum effect. That position is a partial flexion of the hip and full flexion of the knee. This position places the quadriceps under an optimum stretch and still allows full forward extension of the foreleg. Obviously this position is modified by any factor which will modify the tone of the quadriceps extensor muscles, and hence the optimum position of the leg will vary with the tone of the quadriceps.

With respect to the strength of the stimulus, it may be said that within certain limits the stronger the blow to the patellar tendon, the greater the amplitude of the knee jerk. The position at which the hammer strikes the tendon is also important. The hammer should strike the tendon in such a manner as to produce the maximum stretch of the quadriceps.

Among the factors influencing the extent of the knee jerk which have not been given due consideration is the one of mechanical restraint by contraction of the hamstrings, thus interfering with the forward progression of the leg. Secondarily, this also interferes reflexly through the nerve center controlling the knee jerk as a consequence of the reciprocal inhibition of this group of antagonistic muscles.

In a great number of our experiments, respiration modified the extent of the knee jerk. An inspiratory movement accompanied by shivering of the animal increased the tension on the quadriceps through the skin and abdominal muscles. If this occurred at the proper moment, it enhanced the tonus and tension on the quadriceps and resulted in an increased jerk. Any increase in tonus itself will be followed by a reinforcement of the jerk.[4]

Stimulation of the central end of the homolateral sciatic nerve, which causes increased tension of the hamstring muscles (flexion reflex), will lead not only to a mechanical restraint of the knee jerk

4. Luckhardt, A. B., and Montgomery, M. F.: Studies on Knee Jerk; Effect of Acutely Raised Intracranial and Intraspinal Pressures upon Knee Jerk, Am. J. Physiol. **90**:437 (Dec.) 1929.

but also to a reflex inhibition of the quadriceps, that is, if the stimulus occurs just a fraction of a second before the hammer strikes the patellar tendon. Any stimulus which causes reflex contraction of the quadriceps will cause an increased amplitude of the knee jerk.

SUMMARY AND CONCLUSIONS

Stimulation of abdominal viscera may cause:

1. An inhibition of the knee jerk by causing a reflex contraction of the hamstrings, which thus mechanically interferes with the forward movement of the foreleg. Associated with this is a reflex inhibition of the quadriceps due to reciprocal inhibition of the quadriceps. It is also conceivable that stimulation of the viscus may exert a direct inhibitory action on the nerve center controlling the knee jerk.

2. An augmentation of the jerk, especially if the stimulation is very light or if the animal is lightly anesthetized. Under these conditions, we feel that the results can adequately be explained on the same basis as can other sensory stimuli producing an increased tone of the quadriceps muscle which always reinforces the knee jerk. In fact, such stimuli sometimes causes visible extension of the leg.

3. No effect on the knee jerk. This we attribute to deep anesthesia; for on stimulation of the viscera in some animals, we could produce augmentation, inhibition or no effect by merely varying the depth of the anesthesia.

The results were not modified by transecting the spinal cord in the lower thoracic region and thus eliminating the higher centers that might have been involved in the observed reactions.

Clinical Notes

MYELITIS OCCURRING IN THE PRIMARY AND IN THE SECONDARY STAGE OF SYPHILIS*

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Acute myelitis is rarely seen in early syphilis; therefore, when it occurs immediately after treatment with the arsenicals, the possibility of a toxic involvement of the spinal cord as well as a syphilitic myelitis must be considered.

Involvement of the nervous system in the early stages of syphilis has been reported by various authors. Wile and Stokes[1] found that the spinal fluid in primary or secondary syphilis frequently showed a lymphocytosis, increased albumin and globulin content and a positive complement-fixation test. These observations were not accompanied by paralysis and indicated merely meningeal irritation.

Reactions to arsphenamine affecting the nervous system manifest themselves in various forms. Morton[2] classified them as follows:

Nitritoid Crisis. This condition is a manifestation of anaphylaxis occurring during the administration of the drug.

Arsenical Poisoning. The symptoms of this condition occur within a few hours after the administration of arsphenamine and consist of a chill, a rise in temperature and vomiting. The urine is scanty and contains blood and albumin. The patient usually recovers, but the poisoning may be so intense that death ensues.

Serous Apoplexy of Milian. After the injection of arsphenamine, the patient falls into coma on the third day, often without premonitory symptoms, and dies within a few hours. Autopsy reveals edema and congestion of the brain.

Herxheimer's Reaction. This condition is explained by a sudden killing of the spirochetes with a consequent loosening of their toxins. This reaction is manifested by swelling, edema, dermatitis and fever.

In Herxheimer's reaction, the effect on the nervous system is usually manifested by the occurrence of headache, convulsions and coma, in addition to the other symptoms mentioned. No actual paralysis occurs, although there may be intense prostration and weakness. The nitritoid crises may cause similar symptoms. Arsenical poisoning usually gives more evidence of its effects on the peripheral than on the central nervous system. The so-called "serous apoplexy" involves the brain, but there is every reason to suppose that in some cases the spinal cord might be affected with the same congestions and edema and thereby give the clinical picture of an acute myelitis.

The case reported is one of myelitis following arsenical treatment for primary syphilis. It is of special interest because: (1) it is necessary to determine whether

* Submitted for publication, Nov. 11, 1930.

* From the Department of Neurology, University of Michigan Hospital.

1. Wile, Udo J., and Stokes, John H.: Involvement of Nervous System During Primary Stage of Syphilis, J. A. M. A. **64**:979 (March 20) 1915; A Study of the Spinal Fluid with Reference to Involvement of the Nervous System in Secondary Syphilis, J. Cutan. Dis. **32**:607 (Sept.) 1914.

2. Morton, Henry H.: Genito-Urinary Disease and Syphilis, ed. 5, New York, Physicians and Surgeons Book Company, 1924.

the paralysis was due to the syphilitic infection or to arsphenamine in order to know whether or not the treatment should be continued, and (2) it is an instance of actual organic involvement of the spinal cord by syphilis in the primary stage.

REPORT OF CASE

History.—J. J., a man, aged 24, was admitted to the department of neurology at the University Hospital on Nov. 25, 1929, complaining of paralysis of the legs, a penile chancre, incontinence of urine and feces, sore throat and pain in the chest. The family and past history was irrelevant. The patient had worked in a paint shop making castings, thereby coming in contact with various alkalis, zinc, tin and sheet metal.

Three weeks before admission, he noticed a penile lesion which followed exposure two weeks earlier. He consulted his physician, who made a diagnosis of primary syphilis. The Kahn reaction of the blood was 1+. On Nov. 4, 1929, he received 0.6 Gm. of neoarsphenamine intravenously. Following this he was confused and nauseated, and he vomited. One week later, he received 0.9 Gm. of neoarsphenamine intravenously and had a similar reaction. The following day he had a sore throat and a generalized eruption. That evening the right leg was paralyzed, and the following morning he was unable to move either limb. At this time he complained of numbness and tingling in the legs and incontinence of urine. There was no history of pain in the back or the lower extremities. Four days later he came to the University Hospital.

Examination.—The patient was oriented as to time and place and did not appear to be acutely ill. His temperature was 98 F., the pulse rate 82, and respiration 20. The pupils were equal and regular and reacted to light and in accommodation. There was nystagmus on lateral deviation of the eyes both to right and left. There were no palsies of the cranial nerves. The tonsils were small and septic. The biceps and triceps reflexes were equal and normal. Dynamometer readings on the right were 40 and on the left 50. Examination of the heart and lungs showed nothing abnormal. The umbilical reflex was not obtained. The urinary bladder was markedly distended. There was a flaccid paralysis of the lower extremities without any atrophy or deformity. The knee, achilles and plantar reflexes were absent. The vibratory sense was preserved in the ankles. The sense of motion and of the position of the toes was not impaired. There was loss of pain and tactile sensation in the legs and trunk extending up to the sixth thoracic segment. The patient was unable to detect sensations of heat or cold below the tenth dorsal spinal segment. Sensation to pressure was normal over the entire body. The genitalia were normal except in the sulcus coronarius on the right side, where there was a residual induration. The axillary and inguinal lymph glands were of the size of a bean, discrete and not tender. The left epitrochlear gland was palpable.

Laboratory Observations.—With the patient in the prone position, a lumbar puncture revealed a spinal fluid pressure of 220 mm. of water. There was no subarachnoid block. The spinal fluid was clear and colorless, with 84 lymphocytes per cubic millimeter. There was a 4+ globulin and a negative Kahn reaction. The colloidal gold curve was 222332100, and the mastic curve, 332110. No organisms were found on direct examination, and the culture showed no growth.

The Kahn reaction of the blood was ++++. Peripheral examination of the blood showed: hemoglobin, 70 per cent; red cells, 4,500,000, and white blood cells, 14,800. Blood cultures showed no growth.

The urine gave an alkaline reaction; the specific gravity was 1.008; the albumin was 2+; a test for sugar gave negative results; the sediment contained from 30 to 40 pus cells and from 10 to 30 red blood cells per high power field, occasional small granular casts and numerous organisms, with cocci predominating.

Course.—The patient was placed on a Bradford frame. The urinary bladder was treated by the Credé method every eight hours. He received large doses of iodides and urinary antiseptics. The condition was complicated by sacral decubiti which responded slowly to treatment.

On December 2, the myostatic reflexes were increased in the lower extremities, and the achilles reflexes were normal. By Jan. 15, 1930, he was able slightly to flex both knees actively, and on January 25 the achilles reflexes were normal. The knee reflexes were present but diminished. The sensory changes were the same as they had been on admission. The patient was discharged to the Eloise Hospital.

At that hospital examinations of the blood and spinal fluid gave negative results. Starting on February 22, he received twelve intramuscular injections of 1 cc. of bismuth salicylate in oil. During this period he received four intravenous injections of 0.3 Gm. and two of 0.6 Gm. of neoarsphenamine. On July 4, ten intramuscular injections of 2 cc. of bismuth sodium tartrate were started. The condition gradually improved.

On October 8, the patient returned to the University Hospital. He walked with the aid of crutches, but was able to walk a few steps without them. He stated that there was a gradual return of sensation in the lower extremities and that the gait was improving. His greatest difficulty was incontinence of urine and feces. On examination, the pupils reacted to light and in accommodation. There was no nystagmus or palsy of the cranial nerves. The biceps and triceps reflexes were increased. Hoffmann's thumb sign was absent. The umbilical reflex was present. There was no atrophy or deformity of the legs. The knee and achilles reflexes were exaggerated. Vibratory sense was present in the ankles. There were positive Babinski, Oppenheim, Schaeffer, Gordon and Chaddock reflexes on both sides. There was a reaction of defense in both legs. Hypalgesia was present up to the sixth dorsal spinal segment. There were spotted areas of thermanesthesia on the legs. There was a small decubitus on the sacrum. The Kahn reaction of the blood was negative.

COMMENT

The myelitis in this case probably was caused by one of two factors, syphilis or neoarsphenamine. Myelitis has been reported as secondary to sore throat, but in this case the sore throat was not severe and probably was caused by a mucous patch. The facts that the patient showed signs of actual and permanent paralysis and that the paralysis improved following a second course of neoarsphenamine suggest that the myelitis was not a toxic manifestation but a syphilitic, inflammatory involvement of the spinal cord.

Obituaries

CHARLES KARSNER MILLS, M.D.
1845-1931

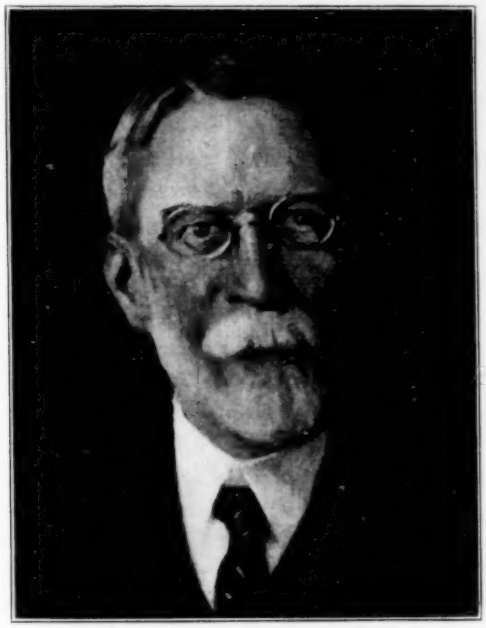
On Thursday morning, May 28, 1931, the second day of the annual meeting of the American Neurological Association, a telegram announced the death of Dr. Mills. On the same morning, a letter was received from Dr. Mills by the secretary of the Association expressing his great regret at not being able to attend. Since his admission to membership in 1881, a period of fifty years, he had missed only one meeting. Two days before his death, in spite of his poor health and protests from his family, he had made all preparations to go to Boston. He died in his eighty-sixth year of an acute edema of the lung following the effort incident to attending a meeting of the Philadelphia Neurological Society about six days before his death. Nothing could more vividly express the dominant motive of his life and the strong will back of it.

Charles Karsner Mills was born at the Falls of Schuylkill, Philadelphia County, Pa., on Dec. 4, 1845, his parents being James and Lavinia Ann (Fitzgerald) Mills. His father was born in Wiltshire, England, and came to this country while a boy; for several generations his mother's ancestors had been residents of the eastern shore of Virginia and Maryland.

Dr. Mills graduated from Central High School in 1864 with the forty-fourth class, known to the alumni of the school as the "War Class" because of the large number of its members who served in the Union Army during the Civil War. He served in the emergency campaigns of 1862 and 1863 in the regiment that was known in Philadelphia as the Second Blue Reserves; in 1862 this regiment was known as the Eighth Regiment, P. V. M., and in 1863, as the Thirty-Third Regiment, P. V. M. The service in 1863, which included the Gettysburg campaign, was very active, the Thirty-Third Regiment being part of the First Brigade of General W. F. Smith's division. When terminating his services as corporal, Dr. Mills had the unique distinction of writing his own discharge, which was signed by his captain, John Dobson.

In a series of articles entitled "The Military History of the Falls of Schuylkill," published in the *Weekly Forecast*, a suburban journal, Dr. Mills gave a complete account of the emergency campaigns of 1862 and 1863 in connection with his historical sketch of the military history of the neighborhood.

For several years after his graduation from high school he was engaged in teaching. He then began the study of medicine, graduating from the medical department of the University of Pennsylvania in 1869. In 1871, he received the degree of Ph.D. After a few years of general practice, his attention was turned to nervous and mental diseases, and in less than ten years following his graduation he devoted his entire time to specializing in this branch of medicine. This was well shown in his publications, which were of general scientific interest, the first being on "Spectrum Analysis," published in 1872. Then followed two papers on the effects of cinchona. In 1875, his first neurologic



CHARLES KARSNER MILLS, M.D.
1845-1931

(From a painting by Hugh H. Breckenridge)

contribution, a "Clinical Note in Forty Cases of Chorea," appeared. From that time on, with the exception of two papers on general medical topics, all of his scientific contributions dealt exclusively with his chosen specialty.

A recital of Dr. Mills' activities in the neurologic field from 1875 until his death would be the history of the progress in neurology in this country, for he was fortunate enough to have been born in the age in which cerebral localization and the specialty of neurology and psychiatry developed. Up to his time there were no neurologists, with the exception of Weir Mitchell, whose work on "War Wounds and

Injuries in the Turner's Lane Hospital, Philadelphia," during the Civil War directed attention on the possibilities in this field.

It was during this period that attention was being focused on neurology by the work of Hughlings Jackson, Hitzig, Ferrier and others. The first meeting of the American Neurological Association was held on June 2, 1875. It was no wonder that Dr. Mills, with his keen insight and brilliancy of mind, chose this specialty, which for him always had such fascination. Realizing that the opportunity to study such patients was necessary, he had himself appointed as neurologist to the Philadelphia Hospital in 1877, this being the first position of its kind in Philadelphia and probably the earliest in the country.

It would hardly be possible to overestimate the influence that the neurologic department of the Philadelphia Hospital has had on the progress of neurology not only in Philadelphia, but in this country. The wards for patients with nervous diseases first occupied a roomy, wooden pavilion and accommodated less than 20 patients. In addition, the wards for paralytic patients were placed in charge of the neurologist. In 1931, the wards contained about 500 patients. It is safe to say that this group of patients, many of whom have been there as long as thirty-five years, represent the finest collection of neurologic material in this country. Much of Dr. Mills' work, as well as that of most of the neurologists of Philadelphia, is based on material from these wards.

In addition, these patients are constantly being used by all the medical schools of Philadelphia, particularly by that of the University of Pennsylvania, which because of its physical proximity, being next door to "Old Blockley," is able to schedule classes for its students in the Philadelphia Hospital without intermission in the school's schedule.

Naturally Dr. Mills was called on to teach at his Alma Mater, and on Feb. 5, 1877, he was appointed lecturer on electrotherapeutics in the medical department of the University of Pennsylavnia. From that time until his death, he was continuously connected with the university in an active teaching capacity. Even before his first teaching appointment, he attended the clinic for nervous diseases in the University Hospital, and in 1874, was made chief of clinic.

Later he was made lecturer on mental and nervous diseases, and in 1893, professor of mental diseases and of medical jurisprudence. In 1901, he was made clinical professor of nervous diseases and in 1903, professor of neurology, which positions he occupied until 1915. After his retirement in 1916, the University of Pennsylvania conferred on him the honorary degree of LL.D., in recognition of his services.

Scientific men, particularly physicians, should be judged by what they have accomplished, not only in their practice but in their teaching,

in their scientific work and in their hospital contacts, and particularly by their influence on others and on their specialty during their lifetime. It would be impossible to think of neurology in this country between 1880 and 1920, a period of forty years, without considering Dr. Mills.

His outstanding characteristic was love of his profession. It mattered little whether it was his hospital work, his teaching or the preparation of his many contributions, for he was interested in anything connected with neurology, and this interest was the great driving force that stimulated those associated with him, and that impelled him to keep on working until his death.

Dr. Mills was particularly happy when he was made professor of neurology at the University of Pennsylvania in 1901 and placed in charge of the department, for up to that time he had had little opportunity to demonstrate what a department of neurology should be. He immediately surrounded himself with a group of the most promising men in Philadelphia, and in a short time, as an evidence of his energy, contributions to the literature made their appearance, and thereafter were published almost annually. So active was his department and so meritorious the contributions that the "Philadelphia School of Neurology" which he created was recognized all over the scientific world. He was a very good teacher, except that he had the habit of discussing the topic in which he was particularly interested perhaps more than the undergraduate relished—a pardonable fault.

Dr. Mills was always interested in the teaching of graduate medical students, for from 1883 until 1898 he was professor of diseases of the mind and of the nervous system in the Philadelphia Polyclinic, of which he was one of the founders. This hospital became a part of the Graduate Medical School of the University of Pennsylvania.

In 1914, with the cooperation of most of the neurologic staff, he organized a graduate course in the wards of the Philadelphia General Hospital. After the foundation of the Graduate Medical School of the University of Pennsylvania, he was made professor of neurology and took active part in the teaching until a few years ago, when he was made emeritus professor.

Dr. Mills' medical activities were not confined solely to the University of Pennsylvania, for from 1891 to 1902 he was professor of nervous diseases in the Women's Medical College of Pennsylvania.

His hospital connections were numerous. Aside from the teaching positions, which have already been mentioned, he was consultant to the Orthopedic Hospital and Infirmary for Nervous Diseases, St. Joseph's Hospital and many others of the state and city hospitals for the insane and for those with chronic nervous diseases in Philadelphia and its vicinity.

Dr. Mills' interest in the American Neurological Association has already been referred to. The first paper that he read before this Association was on "Tumors in the Pons Varolii." From that time until his death he took an active part in its proceedings, always planning a year ahead for the paper that he was to read, usually aiming to present his most important contribution, for he loved the Association, the friends he made in it, the discussions, the annual dinner and everything connected with it. It can safely be said that in its entire history, the Association has never had a more faithful member, nor one who has contributed more to it. He was made president of this organization in 1887 and again in 1924, on the fiftieth anniversary of its founding.

Recognizing the need for a local neurologic association, Dr. Mills founded the Philadelphia Neurological Society in November, 1883. It is hardly necessary to say that he was most active in its discussions and made many important contributions before it. He was made president three times. On the various anniversaries, particularly in his later years, he would delight his audience by humorous reminiscences of the early days of the Society.

Perhaps no one in the field of American neurology has made more contributions to the literature than Dr. Mills. In the semicentennial volume of the American Neurological Association, published in 1924, the list of his contributions takes up eleven pages, more than that of any other member. By actual count, his neurologic contributions alone number 345. It is evident from these that Dr. Mills' predominant interest was in cerebral localization and the philosophy of neurology, to which he has contributed more than any other man in this country. Cerebral localization always fascinated him, although it is obvious that he took no less interest in spinal and neural diseases and in the neuroses. Many of his contributions were in the psychiatric field. Such papers as "Massage and Swedish Movements in the Treatment of Diseases of the Nervous System" and "Galvanism" attest his interest in the practical phase of the subject.

In the realm of cerebral localization, as early as 1888, Dr. Mills insisted on the separation of the motor centers in the cortex from the sensory centers. In conjunction with Dr. Keen and Dr. Frazier, he added to the knowledge of cortical localization by the faradic application to the human cerebrum. He demonstrated, among other things, the center for movements of the head and of the head and eyes and for elements in the movements of Darwin's muscles of grief, the widespread diffusion of the representation of movements of the muscles of the jaw and the differences in stimulation required to produce localized movements by electrical excitation, behind and in front of the

central fissure. In conjunction with others, he indicated the representation of synergic movements in the cerebellum by stimulation of the human cerebellar cortex.

Dr. Mills assumed the existence of an orientation center (higher cerebral vestibular center) in the temporal lobe, and the development of the symptomatology of the parietal lobe may be largely credited to him. He contributed to the knowledge concerning the physiology of taste and uncinate symptomatology.

The record of one case illustrating the localization of the center for word-hearing, with lesions of the superior temporal regions of both hemispheres, has become classic in the history of the subject of aphasia. Dr. Mills made numerous additions to the literature on such subjects as aphasia and the cerebral zone of speech, tumors of the brain and spinal cord, cerebellar symptomatology and localization, cerebral mechanisms and cerebral physiologic topography, the subdivisions of the areas of representation of cutaneous and muscular sensibility and of stereognosis, the subdivisions of the posterior association area of Flechsig designated by him as the concrete memory field, emotional expression and its cerebral representation and the cerebral tonectic apparatus and some of the clinical problems of cerebral tone.

Wilbrand has been credited with first calling attention to macular hemianopia, but, as Dr. W. C. Posey has pointed out, this is due to Dr. Mills.

The recognition of the clinical type known as unilateral ascending paralysis from degeneration of the pyramidal tract is also due to Dr. Mills. On this subject he published not only reports of his original cases, but other papers dealing with different types of both unilateral ascending and unilateral descending paralysis. In 1912, he reported the first case of occlusion of the superior cerebellar artery.

From his bibliography and by a study of his writings, the wide range of subjects covered by Dr. Mills' contributions is apparent, such as neurologic surgery, cerebral morphology, vasomotor and trophic diseases, hydrophobia, multiple neuritis, poliomyelitis, myotonia and athetoid spasm, surface thermometry, electrotherapeutics, problems in electrical potential, hypnotism, hysteria, neurasthenia, psychotherapeutics, mental overwork, Swedish movements and systemized therapeutic exercises, occupational neuroses, localization of tumors of the brain by roentgen exploration, the treatment of aphasia by training, disorders of pantomime (a forerunner of subsequent work on apraxia), the symptomatology of lenticular lesions, intradural root anastomosis, insanity in children and adults, criminal lunacy and the medicolegal aspects of nervous diseases and insanity.

In 1898, there appeared "The Nervous System and Its Diseases," undoubtedly the most scholarly contribution on diseases of the brain and cranial nerves that had appeared up to that time in the American literature. Dr. Mills intended to bring out a second volume, which would include the remaining diseases of the nervous system, insanity and the medical jurisprudence of both mental and nervous diseases, but unfortunately he never did. He also wrote a "Manual on the Nursing and Care of the Nervous and the Insane," a subject in which he was always interested, and early in his career he produced a school book on "Physiology and Hygiene," which was translated into many languages.

Dr. Mills' contributions to medical biography and history have not been without their interest. Among biographical sketches written by him are one on Benjamin Rush and American psychiatry, one on Isaac Ray, the great alienist and medical jurist, and a tribute to his friend of many years, Dr. S. Weir Mitchell. His historical papers include the history of medical jurisprudence in Philadelphia, the history of neurology in Philadelphia from 1874 to 1904 and historical sketches of the Philadelphia Hospital for the Insane and of the Philadelphia Almshouse and Hospital from 1854. One of his favorite recreations was the study of the local history of one of the most interesting sections of Philadelphia, which has for its center the place of his birth, the Falls of Schuylkill. The associations of this old village with the history of the University of Pennsylvania and the military annals of the neighborhood from Colonial times to the end of the Civil War are among his additions to local history.

It is not generally known that Dr. Mills in his early life wrote poetry extensively, although in his later years he disliked to refer to it. His longest poem is called "The Schuylkill, a Centennial Poem by M. K. C.," and was published in book form in 1876 by John Haddock of 104 South Eighth Street, Philadelphia. It contained thirty-four pages with an appendix of fourteen pages and many illustrations of considerable interest.

The field of medical jurisprudence, a subject that he taught in the University of Pennsylvania, always excited his interest. He was an excellent witness, for he had that directness of speech that comes from knowledge of the subject, and his services were largely sought, not only in Philadelphia, but throughout the country. As would be expected, he was called on in many important cases. He made a thorough study of the case of Guitteau both before and after his trial and execution, and was present and took part in the necropsy. He was a witness in the Thaw case.

Dr. Mills was a member of a great many medical societies and held many positions of honor. Besides those that have already been mentioned, he was a corresponding member of the Gesellschaft Deutscher Nervenaertze and of the Société de neurologie de Paris; he held an honorary membership in the Dutch Society of Psychiatry and Neurology, and in the Section of Neurology of the Royal Society of Medicine. He was a past president of the Northern Medical Association of Philadelphia, the Philadelphia County Medical Society, the Medical Club of Philadelphia, chairman of the Section of Nervous and Mental Diseases of the American Medical Association, a fellow of the College of Physicians of Philadelphia and a member of many other societies.

That his interests were not altogether confined to medical matters is attested by the fact that he was a member of the Pennsylvania Historical Society, the City Historical Society of Philadelphia and the Academy of Natural Sciences of Philadelphia, to which he was an active contributor. For many years he was secretary of the Wagner Free Institute of Science, and frequently lectured there.

On his graduation from Central High School in 1864, Dr. Mills began to keep a scrap book. Fortunately he kept this up, and the thirteen books record not only his own progress, but the history of neurology from that time until the death of his friend, Dr. Francis X. Dercum, which occurred about a month before his own demise. His daughter, with loving care, completed the record with the accounts of his death and funeral.

Dr. Mills had always planned to write an autobiography. During his active teaching period and because of his large practice, for his advice was sought all over the country, he had little time to devote to literary pursuits, but in the last ten years he was actively engaged in the preparation of his biography. He had almost completed it and had worked on it in April of this year, expecting to bring it out shortly, as he had already interviewed a publisher regarding it.

Dr. Mills married Clara Elizabeth, daughter of Charles Wilson and Harriet (Friel) Peale of Philadelphia, on Nov. 6, 1873. He is survived by a daughter and three sons.

Perhaps the best criterion of life's accomplishment is in what one does for others. It can be safely said that Dr. Mills developed more neurologists than any one in his time. His enthusiasm was such that no budding neurologist failed to be stimulated by even the most casual contact. It is small wonder that the younger neurologists loved him, for he was a delightful companion, a loyal friend and a wise counsellor. He never failed to advance the interests of others, and if he

thought that their cause was just, he fought their battles with a vigor difficult to imagine in so modest a man.

Endowed with an extraordinary memory, he enjoyed battling for his convictions. He was just in his appreciation of the merit of the work of his confrères, for he had that mark of a great man, a generous enthusiasm and admiration for others.

Surrounded by his family and the books he loved, with the memory of work well done, his declining years were pleasantly and usefully spent, for fortunately he retained his mental vigor until the end. Thus ended the life of one of the outstanding neurologists of America.

THEODORE H. WEISENBURG, M.D.

WILLIAM NORTON BULLARD, M.D.

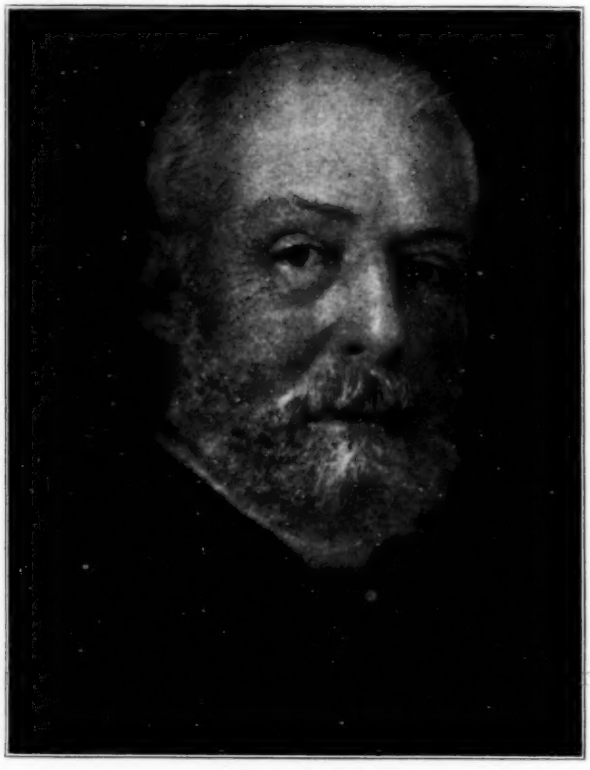
1853-1931

After some years of invalidism, Dr. William N. Bullard died suddenly on April 13, 1931, at the age of 77, at the Ritz Carlton Hotel in Boston, where he happened to be spending a few days.

He was born on Aug. 23, 1853, in Newport, R. I., the son of William Story Bullard, an able financier, and Louisa (Norton) Bullard, a sister of Prof. Charles Eliot Norton. His preliminary education was obtained in various Boston schools, and although he was prepared for college at the age of 17, an attack of typhoid fever necessitated postponement of entrance for a year, which was spent in European travel. Returning to Boston, he was matriculated at Harvard College in the class of 1875, and was graduated from the Harvard Medical School in 1880. After two years as house pupil at the Massachusetts General Hospital, he returned to Europe for a year and continued his studies, chiefly in Vienna. After this thoroughgoing training, he began the practice of medicine in Boston, paying particular attention to neurology. He was soon appointed physician to the Boston Dispensary, and in 1886 assistant to the physician for diseases of the nervous system at the Boston City Hospital, three years thereafter being promoted to the post of physician in the same department. He was closely identified with this hospital for many years, being associated with the late Dr. Morton Prince and Dr. Philip C. Knapp. During those years, there was a service of nervous and mental diseases at the hospital, which it was hoped might develop into a department provided with an adequate number of beds devoted to the care of neurologic patients. Dr. Bullard and others were greatly disappointed when this was abolished, and the hope of a bed service was postponed for many years to the distinct detriment of the development of neurology. Never keenly interested in the actual practice of medicine, Dr. Bullard resigned from his service in November, 1906. During this period he was also connected with the Carney Hospital in South Boston, where much of his clinical work was done. In 1888 he was appointed neurologist at the Children's Hospital, but he gave up this service in 1913 because of impaired health. During the twenty-five years of his connections with this hospital, he was unsparing of his time and devoted to the welfare of the patients under his care, and developed the keen interest which he always maintained in the problems that children presented, especially in relation to feeblemindedness and epilepsy. He was conspicuous in the foundation of the State Hospital for Epileptics at Monson, and became chairman

of the board of trustees. Owing in no inconsiderable measure to his enthusiasm and convictions, this institution, with small beginnings, has developed into one of the most important hospitals of its type in the country and has become an outstanding center for the study and care of this large class of defectives.

Always interested in the furtherance of medical education, particularly as related to the nervous system, in 1909 he established a pro-



WILLIAM NORTON BULLARD, M.D.
1853-1931

fessorship of neuropathology at the Harvard Medical School which properly was termed the Bullard Professorship. The first incumbent of this chair was the late Dr. Elmer E. Southard, who did much toward demonstrating the vital importance of this branch of learning. Recognizing as he did that a delimitation of scope was unadvisable in the rapidly changing state of neurologic knowledge, it was largely due to Dr. Southard that the terms under which this professorship was established were so liberal that its functions embrace the whole field of neurology and psychiatry.

Dr. Bullard retired from active practice in 1912, but continued for some years thereafter in his hospital work, and up to the time of his death never lost contact with medical matters and the furtherance of beneficent medical activities.

Fortunately, Dr. Bullard had the means to gratify his absorbing interest in the collection of books, which he prosecuted with discrimination and judgment for many years. At the beginning he was chiefly interested in sixteenth and seventeenth century classic texts, but gradually he began to buy incunabula and Spanish imprints. Four years ago, he decided to limit his buying entirely to books printed preceding the sixteenth century. In the spring of 1927, Dr. Bullard lent 100 volumes of incunabula to the Boston Medical Library. During the following years he added constantly to this collection, until at the time of his death it numbered 217 titles, in addition to 15 incunabula which he had previously given outright. A catalog of these invaluable books has been compiled by Mr. James F. Ballard, director of the library, printed at the Merrymount Press. Dr. Bullard was not particularly interested in first editions, but he had planned to bring together one edition of all medical books obtainable printed in the fifteenth century, as well as others on collateral subjects. He also collected dictionaries and vocabularies, believing that in such a collection these were essential for the permanent use of scholars. An attempt was made to assemble books such as might have been used for reference at medical institutions in the fifteenth century. In 1928, he presented to the library his sixteenth and seventeenth century books, numbering about 500 volumes, which contained many important classic texts, rare books on witchcraft, a number of Spanish imprints and other outstanding items. Through his will the entire collection of medical incunabula, as well as all his other medical books, ancient and modern, reverts to the library, to which he has also made a bequest of $50,000 to be used for the purchase of books and manuscripts prior to 1700. Among the particularly valuable volumes in the collection are the 1491 edition of John of Ketham's "Fasciculus Medicinae," which is the first medical book illustrated with wood cuts and the only copy in the United States, the "Golden Harps of Nider" printed at Augsburg in 1472, an item of great rarity, a first edition of Holbein's "Dance of Death," Josselyn's "New England Rarities," and Bullein's "Bulwarke of Defence." Arabian authors are also well represented, as well as authors of the twelfth, thirteenth, fourteenth and especially of the fifteenth century, together with books on astrology, magic, witchcraft, demonology, chiromancy and herbals. So unostentatiously was this great collection made that relatively few knew that for many years Dr. Bullard, ably seconded by his wife, had been laboriously bringing together these literary treasures, which are of great value now,

and which will increase immeasurably in significance in the coming generations.

An avocation of quite different type was his interest in and knowledge of botany, to which he gave much time and study. Both in this country and in Europe he wandered much afield in the observation of wild flowers and plants, and derived therefrom a peculiar pleasure and satisfaction.

During his earlier years, although he never wrote extensively, he published many papers on neurologic subjects, the more important of which related to epilepsy, idiocy and imbecility. His articles and investigations, however, included many on functional and organic neurology and several on problems of surgery of the central nervous system; the latter were done in collaboration with the late Dr. H. L. Burrell. A notable contribution made in 1886-1887, which attracted much attention at the time, was on the subject of chronic tea poisoning, in which Dr. Bullard attempted with considerable success to show that a certain fairly well defined symptom complex might be induced by overindulgence in tea. The papers that he published on this subject were based on the careful observation and study of nearly 200 cases of confirmed tea drinkers.

In regard to his connections with societies, he took particular interest in the National Society for the Study of the Feebleminded and the National Association for the Care and Study of Epilepsy, of both of which he was president at one time. He was likewise president of the American Neurological Association on the occasion of its meeting in Boston in 1912. Unfortunately he was not able to preside, since at the time of the meeting he underwent a serious operation which nearly cost him his life. He was a member of many local societies, but for many years had been an inconstant attendant, possibly owing to his decline of interest in the medical matters under discussion and also, in his later years, to the necessity of conserving his strength. He held membership in the Somerset Club and was one of the founders of the Tavern Club, which has always held a peculiarly unique position in the literary and artistic life of Boston.

For approximately six years preceding his death, he was afflicted with a serious disorder of the heart, which was ultimately fatal. These years were furthermore clouded by failing eyesight, so that toward the end he was practically blind. In spite of these physical handicaps, he maintained his interest in life and in his special avocations and still found opportunity to continue with enthusiasm the various tasks that he set himself. He was an omnivorous reader, and in spite of failing eyesight his determination enabled him to derive satisfaction in this resource nearly to the end. For the greater part of his life he was closely iden-

tified with Boston, but for some time past he had made his residence at Lenox and traveled much abroad and in this country. He spent his summers at North Haven, Me.

As a man, Dr. Bullard had strong personal convictions, but he was genial, sympathetic and helpful to those who sought his advice and counsel. As a diagnostician, he was skilful and possessed of peculiarly sound judgment in the broader aspects of medicine, as well as in the special field of the nervous system. As a citizen, he was public-spirited, devoted to good works and eager to promote all worthy causes. His life was characterized by extreme modesty, and he had an unquenchable desire to assist younger men toward the accomplishment of their ambitions and to further the advancement of medical science and practice. The establishment of a chair of neuropathology demonstrated his recognition of the need of encouragement of research and the development of opportunities for young men in this important field, which at that time was little recognized as a fundamental discipline. The liberal endowment of this chair has made possible a noteworthy development of neuropathology in its widest sense in Boston and in the broader sphere of American medicine in general. It was doubtless, therefore, a great satisfaction to him that the trustees of the Boston City Hospital approved the recommendation of Dr. Stanley Cobb, second Bullard professor, that the upper floors of the new medical building of that institution be named the Bullard Neurological Laboratories; happily, he lived to see this recommendation carried out and the laboratories in active operation.

Although for many years inactive in a purely professional way, Dr. Bullard never lost interest in neurology and its needs. He will be remembered in succeeding generations as one of its earlier and most active pioneers in bringing it to the position which it now holds among the special branches of medicine.

Rather late in life he married Miss Mary R. Reynolds, who survives him; she is a sister of Dr. Edward Reynolds. They had no children, and with his death the male descent of his family comes to an end.

E. W. Taylor.

Abstracts from Current Literature

THE LOCALIZATION OF THE VESTIBULAR REACTION OF THE EXTENDED ARMS AND OF THE POINTING TEST; THE DIAGNOSTIC VALUE OF THESE REACTIONS IN THE COURSE OF DISEASES OF THE CEREBELLAR FOSSA. G. S. ZIMMERMANN and A. S. TCHERNYCHEW, Rev. d'oto-neuro-opht. **8**:503 (July) 1930.

The anatomic and physiologic relations between the labyrinth, the vestibular system and the cerebellum are of the highest importance for otologists, neuropathologists and neurosurgeons. Bárány's ideas of the mechanism of the vestibular reactions in the pointing tests have been contested by numerous writers. In the present article the authors give an anatomicoclinical exposé of one of their cases and discuss the question of these reactions. The diagnostic value of the pointing test in lesions of the cerebellar fossa is formulated from a study of twenty-six cases of such lesions observed by the authors.

A girl, aged 16, entered the clinic on Dec. 15, 1927, with a history of headaches, intermittent vomiting, vertigo, loss of vision, pain at the junction of the occiput and the neck and tinnitus. Her father was an alcoholic addict. Six months previous to admission, pains appeared in the left occipital region, occurring every few days. They became more frequent; vomiting and vertigo set in, and, during the previous two and a half months, vision had rapidly failed. A neurologic examination revealed: lessened perception of odors in the left nostril; hyperalgia in the domain of the superior branch of the left trigeminus; displacement of the lower jaw to the left when the mouth was closed; deviation of the tongue to the left; lessened grip in the left hand; oscillation without definite direction in the Romberg test; inclination of the head to the right; a vacillating gait with the legs wide apart; slight incoordination on the right in the finger-to-nose test. The conjunctival reflexes were absent, and the corneal reflex was lively on the right side. The patella and the achilles reflexes were absent. The Babinski and the Mendel-Bechterew signs were present on the left. The cerebrospinal fluid showed no pathologic modifications. Examination showed also: bilateral amaurosis with perception of light only, mydriasis, equal and round pupils, absence of the reactions to light and in accommodation, atrophy of both optic nerves following papillary stasis, and exophthalmos of the right eye. An examination of the blood showed 80 per cent lymphocytes, 17 per cent eosinophils and many transitional forms. The Wassermann test gave negative results for the blood and the spinal fluid. The whispered voice was a little less audible in the left than in the right ear; in the Weber test the sound was lateralized to the right and bone conduction was shortened. The neuro-otologic diagnosis was a lesion probably in the left cerebellar fossa. Five weeks later, an examination in the surgical clinic revealed the following: When lying on the back, the patient showed a spontaneous horizontal nystagmus of large amplitude toward both sides, provoked by a minimal lateralization of the eyes; when lying on the right side, there was nystagmus which was accentuated on looking toward the left; when lying on the left side, there was nystagmus to the right on looking straight forward; when lying on the abdomen, there was no nystagmus; there was no past pointing or deviation of the arms in the vertical or horizontal positions. After douching the right ear with 5 cc. of water with the head back, there was horizontal nystagmus to the left; ten minutes later, a spontaneous nystagmus to the right appeared. Douching the left ear caused a lively horizontal nystagmus to the right. The hands deviated normally. A diagnosis of a lesion in the right cerebellar fossa was made.

An operation on the skull was undertaken but was not completed. Death occurred two days later.

At autopsy a hemiangio-endothelioma, measuring 2.5 by 1.5 cm., was found in the superior semilunar and in the posterior quadrilateral lobes of the right cerebellar hemisphere, directly to the right of the superior vermis. The cortex of the affected lobes in contact with the tumor was much changed: the molecular layer was thin, resembling a limiting membrane soldered to the tumor tissue; the ganglionic elements had almost disappeared. There were few Purkinje cells, and they were far apart, changed in shape and small. Some of them were cornified into small balls, deeply stained. The granular layer in immediate contact with the tumor was contracted and rarefied, and it contained quantities of calcareous deposits. The stellate and horizontal elements had disappeared.

On sagittal section a cyst was found in the white substance of the right hemisphere. It was developed in a dorsoventral direction and was narrow at its origin, but became larger until it attained a diameter of from 0.4 to 0.5 cm. It communicated with the fourth ventricle, but was independent of the tumor. It was surrounded by a band of rarefied and altered fibers. The parts of the cerebellum that were adjacent to the cyst (the anterior and posterior quadrilateral lobes) and that formed its superior wall were compressed, and the subjacent white substance was rarefied. This rarefaction extended to the white substance of the biventer lobe and to part of the lobus gracilis, but the cortex here was not compressed. At the level of the dentate nucleus, the cyst was in the white substance and compressed the sublobar stratum, the anterior quadrilateral lobe and, partly, the tonsil. It did not extend to the vermis, but the latter, especially at the level of the uvula, the nodulus and the median posterior lobe, showed retraction of the cortical layer. In the cortex adjacent to the cyst the alterations were less marked than in the cortex that was in contact with the tumor. The molecular layer was a little retracted and contained many newly formed vessels. The basket cells were relatively well preserved. In the intermediate layer the disappearance of Purkinje cells was less accentuated. The granular layer was not rarefied, but the white fibers and the supraganglionic, the infraganglionic and the intraganglionic plexuses were rarefied. The climbing fibers were poorly delimited. In the cortex of the remainder of the right hemisphere there was a more or less advanced necrobiotic process, but it was less intense than that previously described.

The dentate nucleus preserved its form and volume. The ganglion cells of this nucleus and of the nuclei globus and emboliformis were modified in form and volume. Cornified and pyknotic elements, homogeneous and shadow cells, were often found in conjunction with marked glial proliferation. In the cortex of the left side of the vermis, the degeneration and atrophy of the cellular elements, accompanied by glial reaction, resembled that found on the right. In the cells of the nuclei of the roof a necrobiotic process in various stages, accompanied by glial proliferation and a thickening of the fibers of the glioreticulum, was found. A certain number of changes in the cells of the bulb similar to those already described, especially in the left inferior olive, were present. There were no special changes found in the pons.

The otoneurologic symptoms were: inclination of the head toward the right shoulder, spontaneous outward past pointing of the left arm, absence of reactions from the vertical canals of the right side, hyperexcitability of the horizontal semicircular canals on douching the ears, absence of the falling reaction backward after douching the ears, change in the intensity of the spontaneous nystagmus in the various positions of the head and trunk, change in the direction of the nystagmus ten minutes after the caloric test, and normal past pointing after rotation and douching; all occurred in a patient with a lesion of the right side of the cerebellum.

In the present discussion, only one observation is analyzed: normal deviation in the pointing test and the deviation of the arms in the course of the vestibular examination. The analysis is supported by the observations in twenty-five other cases of disease in the posterior fossa, and the diagnosic importance of the deviation is emphasized.

The conception of Bárány that the mechanism of spontaneous deviation and deviation after vestibular stimulation is located in the cerebellum is not accepted.

Bárány's hypothesis was based on the observations of a number of investigators (Adamkiewicz, Rottmann, Bolk, Edinger, André-Thomas) and on his own chilling experiments. His theory was supported by Kohnstamm, who found that the vestibular nerve does not go directly to Deiters' nucleus but that a vestibular fiber, coming from the cerebellum, terminates there and that there are pathways going to the anterior horns of the spinal marrow that take their origin in Deiters' nucleus. Bárány's clinical material consisted of twenty cases, of which only five came to autopsy. Only macroscopic examinations were made in these cases. A description of Bárány's pointing centers is given.

DENNIS, Colorado Springs, Colo.

THE LOCALIZATION OF THE VESTIBULAR REACTION OF THE EXTENDED ARMS AND OF THE POINTING TEST: THE DIAGNOSTIC VALUE OF THESE REACTIONS IN THE COURSE OF DISEASES OF THE CEREBELLAR FOSSA. G. S. ZIMMERMANN and A. S. TCHERNYCHEW, Rev. d'oto-neuro-opht. **8**:583 (Oct.) 1930.

In a case of tumor of the right side of the fourth ventricle, published in 1922, the right hand could not be made to past point to the left. This was thought to be due to hypofunctioning of the vestibular apparatus and not to involvement of Bárány's centers. The information gained from the case reported here will explain this phenomenon. In 1923, Barré repeated Bárány's chilling experiments. He believed that the thermic action was transmitted through the cerebellar cortex to the labyrinth by a vasomotor reflex. His conclusions were: 1. Past pointing is a vestibular, not a cerebellar, reaction. 2. Bárány's theory of cerebellar localization must be abandoned and his chilling experiments must be otherwise explained. Barré observed in a series of cases of cerebellar lesions that the elements of the labyrinthine syndrome, which are present early and frequently in these cases, differ from those seen in pure labyrinthine disease. These anomalies are: lack of past pointing with good nystagmus, deviation of the arms and nystagmus to one side and deviation of the trunk to the opposite side. In all the cases with anomalies of the vestibular syndrome Barré found other cerebellar signs: dysmetria and adiadokokinesis. He concluded: 1. Disturbance of the cerebellar function may be manifested by disturbances of the normal vestibular syndrome. 2. When there is disharmony in the vestibular syndrome, a cerebellar factor must be looked for as the cause of it. The authors are in complete agreement with this view.

Bárány attributed a cerebellar localizing value to these anomalies, and almost all writers believe that an examination of the vestibular function gives information about the state of the cerebellum. This is not always true, because an anomalous vestibular reaction may be caused by a distant condition acting on the cerebellum.

De Stella expressed the belief that the functioning of the labyrinth is independent of the cerebellum. In his case of disease of the right cerebellopontile angle, after extirpation of the tumor the hearing and the vestibular functions were reestablished and all disturbances of the cranial nerves disappeared, although the right cerebellar hemisphere was removed. The question arises, "What purpose is served by the pathway (cerebellovestibular tract) that connects the cerebellum and the vestibular apparatus?" Stenver's theory was that the cerebellum constitutes a supplementary reflex pathway of the vestibular apparatus and that the development of this pathway is the result of long training. He based his theory on the facts cited by Edinger, who traced the evolution of the cerebellum from the mixines, which have no cerebellum, up through fishes and swimming birds. Destruction of these pathways causes the disappearance of the acquired functions: precision, harmony and perfection of movements. The case of Fischer and Pötzl, in which a large part of the right cerebellar hemisphere was removed, led them to believe that the cerebellum is not indispensable to the production of tonic vestibular reactions. However, the absence of its regulator function results in disharmony of the vestibular reactions.

Sven Ingvar expressed the belief that there is not a distribution of function in the cerebellum according to the parts of the body, but according to different directions of movement and falling; the cerebellum receives and transforms the unconscious sensation of mass and, by reflex action, it counteracts the impressions of the force of gravity and inertia in the body and thus preserves equilibrium.

The mechanism of past pointing is complex. When a person is asked to move the arm in a certain way, he must sense the direction, thus bringing the cerebral cortex into play. When different parts of the cerebrum are chilled, if the center of sensibility of direction is not touched, the subject will point correctly. The terminal cortical vestibular neuron probably ends at this point. When the excitation of this neuron passes a certain degree (rotation and caloric tests), the normal sensation of direction is disturbed and the efferent impulse to the shoulder is not precise; at the same time the impulse from the stimulation is directed to the subcortical nuclei, especially those of the bulb and pons, and only partially to those of the cerebellum. These subcortical nuclei are directly connected with the anterior horns of the cord by the vestibulospinal pathway and thus deviation of the extremities is produced. The central mechanism of past pointing comprises: (1) increase of tonus of the extremities in consequence of excitation of the subcortical centers from vestibular stimulation; (2) modification of the cortical impulse because the movement is sensed. At times the cortical innervation is greater; at other times, the subcortical. The predominance of one or the other of these innervations is revealed by the absence or the presence of deviation in the pointing test. This explains the different results obtained when using the pointing test in normal persons.

In the case reported, the right cerebellar center for tonus of the movement of the arm downward was destroyed, the subcortical systems having been particularly injured, but the subcortical vestibular pathways in the pons and bulb were intact. Hence, the conclusion is evident that the reaction and the deviation of the right arm passed through the following reflex arc: vestibular nerve, nuclei of the bulb and pons, vestibulospinal pathway, anterior horns of the spinal marrow; not, as Bárány assumed, to cerebellar cortex, central cerebellar nuclei, red nucleus and Monakow's bundle.

While adhering to the doctrine that vestibular deviation of the arm is not a cerebellar function, it is recognized that the cerebellum, through its anatomic connections with the nuclei of the vestibular apparatus and with the cerebral cortex of the opposite side, may change the aspect of vestibular deviation. And, as the muscles of the trunk and the extremities are constantly subject to labyrinthine tonus, labyrinthine and retrolabyrinthine diseases that affect the bulb, vestibulocerebellar pathways or the frontal or temporal cerebral regions may provoke spontaneous deviation of the arms; and, also, either directly or by acting from a distance, interference with any one of the elements of the reflex arc must cause deviation.

The antomic hypotheses formulated by Ramón y Cajal and Kohnstamm, which supported Bárány's theory of localizations in the cerebellar cortex, are not confirmed by the recent researches of André-Thomas. According to the latter, only a few fibers of the root of the vestibular nerve go directly to the nuclei of the roof; a larger number originate in the nuclei of the vestibular nerve; the rare fibers of the bulb and pons are directed toward the cerebellar nuclei and enter the direct sensory pathway; the fibers of the vestibular nuclei go to the cerebellum and leave it as direct or crossed fibers. The uncinate tract (Russel) and the tectobulbar bundle (van Gehuchten) also unite the nuclei of the roof to the nuclei of the vestibular nerve. In spite of the intricacies of the two systems, it is possible to isolate vestibular from cerebellar symptoms. Labyrinthine spontaneous deviation (peripheral) is always accompanied by other labyrinthine symptoms, such as vertigo and horizontal nystagmus of small amplitude, but never by intracranial hypertension; it is habitually bilateral and is directed toward the slow phase of nystagmus; at times, it is unilateral but if so, there is a clear difference in the excitability of the two labyrinths. Spontaneous deviation of vestibular

(retrolabyrinthine) origin is accompanied by an incomplete vestibular syndrome: labyrinthine vertigo is not always present, nystagmus is of wide amplitude and is more lively, and in most cases the deviation is directed toward the side of the lesion. When the lesion involves a considerable part of the cerebellum, especially the subcortical region, the vestibular syndrome is accompanied by typical cerebellar symptoms, such as ataxia, astasia, bradyteleocinesia or adiadokokinesis.

When the lesion is in the pathways of the middle or anterior cerebral fossae, vestibular symptoms are usually lacking. At times on stimulation (rotatory, caloric) the rapid phase of nystagmus is absent, and spontaneous deviation of the arm is toward the side opposite the lesion. These observations are supported by the results of Groebell's experiments on pigeons.

On this basis the outward deviation of the left arm at the first examination in the case reported may be explained. According to Goldstein, cerebellar deviation is the result of the tendency to the movements of extension and abduction, which are automatic tendencies. Total removal of the cerebellum causes extension and abduction of the lower limbs; irritation causes adduction and flexion. This is also true of the cerebrum. Hence, the cerebrum and cerebellum inhibit automatic movements, and when this inhibition is removed by disease, spontaneous deviation is observed. Goldstein's ideas are confirmed by the authors' twenty-six cases. Of eleven cases with a lesion in the cerebellopontile angle, spontaneous deviation was absent in six and present in five. Of nine cases with tumor of the cerebellum, deviation was present in five, doubtful in one and absent in three. In one case of abscess of the angle and in one of meningitis of the cerebellar fossa, spontaneous deviation occurred. In the twenty-six cases there were fifteen with spontaneous deviation (58 per cent). Of these, the deviation was toward the lesion in eight. Contrary to the opinion of many observers, this has some localizing value. Most often it is toward the side of the lesion, according to Aubry. If the spontaneous and vestibular deviation is considered with reference to the seat of the lesion, Bárány's theory will be found in default in many. In the case reported, spontaneous deviation of the left arm outward, together with intracranial hypertension and slight cerebellar symptoms, points to a process acting on the cerebellar nuclei or on the pathways connecting them with the central vestibular system. The function of the regions damaged is to inhibit the reflex system; the right uncinate and the left entoconjunctival tracts are in relation with the left vestibular system. Hence, the important lesion on the right and the lesser one on the left resulted in liberation of the left vestibular apparatus, with the consecutive appearance of automatic movements, i. e., deviation to the left. The disappearance of the spontaneous deviation at the last examination is explained by the compensatory action of the cerebrum. In the case of abscess in the angle, the deviation was upward. According to Bárány's theory, this would indicate a lesion in the posterior median pole of the cerebellum. The authors believe that it indicated disease of the cerebellar function in general and not a lesion of a definite region of the cortex.

In case 17, cold irrigation of the right ear did not reverse the spontaneous outward deviation of the left arm. This is explained as follows: "Disease of the uncinate tract and other pathways in the cerebellum damages inhibitory and reflex functions, causing spontaneous deviation of the index finger and arm in different directions." The stimuli directed to the labyrinth in this case were not powerful enough, or the vestibulospinal reflex was not in condition to overcome the deviation, which was due to abolishment of the inhibitory function of the cerebellum.

In case 7, the right arm did not deviate inward during cold irrigation of the right ear. This is explained by the fact that tumors of the angle cause necrobiosis in the region of Deiters' system (Tchernychew). The reflex arc (from the vestibular nuclei to the vestibulospinal tract) was damaged in this case, causing absence of the reaction.

The authors believe that they have simplified the clinical comprehension of these phenomena, although they admit that they may, in the future, have to supplement or correct the theories advanced here.

DENNIS, Colorado Springs, Colo.

REPORT OF THE MAYOR'S COMMITTEE ON DRUG ADDICTION TO THE COMMISSIONER OF CORRECTION, NEW YORK CITY. ALEXANDER LAMBERT and COMMITTEE, Am. J. Psychiat. **10**:433 (Nov.) 1930.

The mayor's committee on drug addiction studied 318 opium addicts in a special ward at the Bellevue Hospital. Most of these patients had had a public school education; 18 per cent of them had high school diplomas, and 6 per cent had attended college. Most of the subjects were of average intelligence, and physically they showed no evidence of any damage as a result of the addiction. More than half the patients were classified as constitutionally psychopathic. In determining the efficacy of the methods of treatment, the withdrawal symptoms were grouped into four classes—gastro-intestinal, neurologic and muscular symptoms, and symptoms of prostration—and their intensity was gaged on a scale from 0 (indicating absence of symptom) to 4 (indicating excessive manifestation of symptom). As each treatment was given, an equal number of patients received placebos that simulated in color and in other ways the treatment being followed. Neither the nurses nor the patients knew which patient was a control and which was receiving therapy. For instance, the control subjects received a dilute solution of bismarck brown which had the color and general appearance of a certain proprietary preparation and to which enough glycerin had been added to produce the stinging that accompanies injection of this drug.

It was found that untreated patients, that is, those from whom the morphine was withdrawn abruptly, showed withdrawal symptoms within a few hours of the last administration of opium, and within twenty-four hours manifested the severe symptoms of withdrawal; these remained at an intense level for another twenty-four hours, when they began to subside; at the end of seventy-two (or, at the most ninety-six) hours, withdrawal symptoms had largely vanished. The symptoms were grouped and graphed on a scale of intensity and time, and the variations produced by different treatments were similarly charted and compared. When "narcosan" was used, it was found that the gastro-intestinal symptoms were more severe, while the other manifestations were unchanged; there was no evidence that the craving for drugs was diminished after treatment with narcosan. Atropine lessened the diarrhea, but seemed to make prostration more likely, and in general failed to reduce the withdrawal symptoms or obliterate the craving. Hyoscine caused delirium in more than half the patients who received it, but otherwise seemed of no value either in managing the patient during withdrawal or in rehabilitating him afterward. A 50 per cent solution of magnesium sulphate, injected with a little procaine hydrochloride in a dosage of 0.25 Gm. of salt per kilogram of body weight, produced relaxation and definitely diminished the amount of restlessness, but severe local irritation was produced at the site of the injections, and the results did not justify its use. Sodium amytal succeeded in bringing about sleep but, except as a temporary measure in quieting the patient, was without value.

The reduction treatment was studied in four forms, abrupt withdrawal, gradual withdrawal (seven days), slow withdrawal (fourteen days), and rapid reduction with codeine. Abrupt cessation of administering the drug seemed cruel, and often produced collapse; on the other hand, its very severity had a certain psychic effect that made it a valuable method in treating healthy young addicts. In the seven day schedule, the withdrawal symptoms were much less intense than in the cases of abrupt withdrawal, while patients who were placed on the fourteen day program suffered from light withdrawal symptoms. Alternating morphine with codeine during a period of slow reduction produced results similar to the seven day reduction schedule. The committee's conclusions were that there is, as yet, no specific for the treatment of addiction to drugs, nor for the relief of withdrawal pains; that the system of gradual reduction over a fourteen day period is humane and safe, but carries several disadvantages—it is time-consuming, requires great vigilance, and robs the patient of the dramatic effect of severe withdrawal pains, which have a certain symbolic, psychologic value.

The committee could find no satisfactory method of rehabilitating the addict; as far as could be traced, all the patients returned to the use of the drug after

discharge from the hospital. Realizing that most of these subjects were constitutionally inferior, the committee expressed no surprise at this result.

Cocaine is not, at present, much of a problem, as it brings such severe acute mental disorder and physical deterioration that even among the addict classes it is widely shunned. Of the addicts to opium, the majority were enslaved to heroin. The committee refuses to believe that addiction is a disease in the usual sense of the word. The study also shows that most of the addicts fall into this habit through bad association; less than 5 per cent were led into the addiction through injudicious medication prescribed by a physician. Most of the habitués could give no serious reason for falling back into addiction after being freed from the poison by a course of withdrawal treatment.

The committee closes the report with recommendations for better institutional care during withdrawal and intelligent supervision during a probationary afterperiod.

DAVIDSON, Newark, N. J.

HORIZONTAL NYSTAGMUS WITH CHANGING DIRECTION ACCORDING TO THE SUPPRESSION OF THE VISION OF ONE OR THE OTHER EYE: TONIC AND KINETIC DISTURBANCES OF THE EYES FOLLOWING TRAUMATISM IN A PERSON WITH SYPHILIS. D. MANOLESCO, S. DRAGONESCO and D. LAZARESCO, Rev. d'oto-neuro-opht. **8**:606 (Oct.) 1930.

A man, aged 30, came to the clinic because of difficult vision. There was nothing significant in his past history except that his wife had had only one child, which was stillborn. Two years previous to admission, he received a blow on the occipital region and was in a comatose condition for three days. On recovering from the coma, he heard almost nothing in the right ear and his vision was reduced to the perception of light. He improved somewhat, but several days later he accidentally discovered that the right eye was blind. During three months he suffered with left hemicrania, which was worse in the frontal region.

Examination revealed a robust man with normal motility and coordination of the limbs. Hearing in the right ear was much reduced; in the Weber test the sound was not lateralized, the Rinné test gave negative results, and bone conduction was shortened on the right side. The pupils were normal. Visual acuity was reduced to one-half in the left eye and to perception of movements at 30 cm. in the right eye. The visual field in the left eye was contracted; the fundi were normal. Ocular motility was disturbed: with the right eye covered, movements of the left eye were normal; with the left eye covered, there was slight limitation upward and outward in the right eye. There were present: internal strabismus of the right eye; rapid and fine gyratory nystagmus in both eyes and a horizontal nystagmus to the left, which was intensified on looking to the left; inability to turn the right eye completely to the right. When the left eye was covered, a horizontal nystagmus in the right eye to the right appeared, followed by marked internal deviation inward in the right eye; when the right eye was covered, there was a horizontal nystagmus to the left. There was no tonic deviation of the arms or the trunk.

A galvanic test of the ears, with the positive pole on the right, showed the following: with 2 ma. the head inclined to the right; with 4 ma. the arms deviated to the right; with 8 ma. there was a horizontal and gyratory nystagmus to the left. If the right eye was then covered, the nystagmus was increased; if the left eye was covered, the right eye became immobile, but on increasing the current to 12 ma. nystagmus to the left reappeared. With the positive pole on the left side, the same phenomena occurred, but they were reversed.

On irrigation of the right ear with the head backward at an angle of 60 degrees, after 40 cc. of cold water had been given, there was horizontal nystagmus to the left, which changed to the right on covering the left eye; when 130 cc. of water had been used, the nystagmus to the right ceased, and after the use of 160 cc. of water, nystagmus to the left was reestablished. Deviation of the arms occurred following irrigation with 40 cc. of water. During the irrigation both eyes were

converged, the right one markedly. Rotatory nystagmus did not occur when the head was put forward 30 degrees. During irrigation of the left ear, pronounced internal strabismus of the left eye appeared at once; when 100 cc. of water had been used, the spontaneous nystagmus to the left ceased, to occur again feebly on covering the right eye; but on covering the left eye, a marked nystagmus to the right appeared; when 120 cc. of water had been used (both eyes being open), marked horizontal nystagmus to the right was seen; covering neither eye changed this direction. When the head was tilted 30 degrees forward, rotatory nystagmus did not appear. Deviation of the arms was normal.

Rotation to the right caused intense horizontal nystagmus to the left, uninfluenced by covering either eye. Rotation to the left provoked a horizontal nystagmus to the right of lesser intensity, which could be neutralized by covering the right eye. Deviation of the arms was normal. Changing the position of the head and body suddenly on the same plane or turning the head to the right or the left caused a marked convergence of both eyes, followed in one-half minute by a nystagmus to the right.

Examination of the spinal fluid revealed: positive reactions to the Nonne-Apelt, Pandy and Bordet-Wassermann (3 plus) tests; 12.3 cells per cubic millimeter. The Bordet-Wassermann test of the blood was also positive. There were no neurologic signs of disturbances of the other cranial nerves.

Change in the direction of nystagmus and also inversion of nystagmus have been noted by numerous writers. The nystagmus to the right, appearing when the left eye was covered, was of central origin, was intense, was not due to fixation (because it was present even when the good eye was covered) and was not a railroad nystagmus. It is difficult to understand the change of direction of the nystagmus in relation to covering one eye. The deficiency in the movements of the eyes upward and the spasmodic convergence both point to a lesion of the supranuclear pathways, especially the posterior longitudinal bundle. Probably both the traumatism and syphilis are factors in the etiology.

DENNIS, Colorado Springs, Colo.

THE CENTRAL NERVOUS SYSTEM CONTROL OF THE OCULAR MOVEMENTS AND THE DISTURBANCE OF THIS MECHANISM. HENRY ALSOP RILEY, Arch. Ophth. **4**:640 (Nov.) and 885 (Dec.) 1930.

This article is a detailed study of the machinery provided for the ocular movements by the central nervous system and of disturbances in this mechanism. The work is divided into two parts which are published in succeeding numbers of the *Archives of Ophthalmology*.

Part 1 is a consideration of the control of the ocular movements by the extra-ocular muscles, and part 2, disturbances of ocular movement caused by interference with the nuclei and the intra-axial course of the oculomotor nerves.

In considering this article, the reviewer wishes to call attention to the abstract which follows of three articles by Cornelis D. Verrijp of Leiden, Holland: "Ocular Movements" (*Arch. Ophth.* **4**:73 [July] 1930), "On the Analysis of Ocular Movements," communicated by Prof. J. Van Der Noeve, on Feb. 25, 1928, before the Royal Academy of Science in Amsterdam, and "On the Analysis of Ocular Movements," also presented before the Royal Academy of Science in Amsterdam, 1930.

Dr. Riley opens his dissertation by a brief discussion of the action of the extra-ocular muscles and their innervation and of disturbances in this mechanism. Part 1, which considers the control of the ocular movements by the extra-ocular muscles, is divided into the following: (*a*) normal control, (*b*) the disturbances of the control of the ocular movements produced by disease of the extra-ocular muscles, (*c*) the control of the ocular movements by the peripheral oculomotor nerves, (*d*) disturbances of ocular movement produced by interference with the peripheral oculomotor nerves, (*e*) the nuclear control of the oculomotor muscles, (*f*) the brain stem centers controlling the ocular movements and (*g*) the center

for lateral deviation of the eyes, vertical deviation, convergence and divergence and the various reflex movements of the eyeball. In this section there is a good review of the literature, a discussion of the various pathologic studies and good line drawings to describe the various mechanisms and the ocular movements. Brouwer's chart showing the morphologic segmentation and functional localization of the oculomotor nucleus according to various investigators, which is a well known and valuable graphic comparison, is included.

Part 2 opens with a discussion of the various criteria for differentiating between paralysis of the nuclei, root and gaze; the various diseases of the nuclei, neuroaxial centers or the root fibers are then considered and grouped from an etiologic standpoint. The paragraphs on differentiation are as outlined by Wilbrand and Saenger. Weber's, Benedict's, Millard-Gubler's and the Foville syndromes are briefly presented. Hyperkinetic disturbances are discussed as symptoms that are much less common than the hypokinetic or paralytic types of oculomotor dysfunction. Nystagmus and nystagmoid movements are detailed. In connection with the reflex disturbances of the eye, these seem to be the most important.

The control of ocular movements by the extrapyramidal system is, as the author states, "the consideration of a field that is not only difficult on account of its great complexity, but also uncertain because of the indefiniteness of our information in regard to many of the elements that are concerned in the extrapyramidal control of voluntary movement." This includes consideration of the cerebellum, the peduncles of the cerebellum and the so-called striatal system which is controlled by the basal ganglia and associated structures, including the caudate nucleus, the putamen, the globus pallidus, the corpus subthalmicum of Luys, the substantia nigra and the nucleus ruber. The pathology of these parts of the central nervous system as manifested in disturbances of ocular motility are considered. Oculogyric crises (relative to which there has been much literature recently) are reviewed. A discussion of binocular vision from the standpoint of ontogeny and phylogeny is included in its relevancy to disturbances of the coordinate movements of the eyeball, properly considered as the oculogyric movements.

The preservation of fixed gaze, unusual volitional control of the movements of the eyes and disturbances in cortical control of the ocular movements, as well as functional disturbances of oculomotor control, are discussed in the conclusion.

The content of anatomy, physiology and pathology covered in this article is too extensive to be more than suggested here. The illustrations are all most valuable. Pathologic changes are detailed satisfactorily. The structure of the paper is comprehensive and exhaustive. References to the literature on the subject are frequent and made valuable by complete footnotes. The reviewer recommends strongly a careful perusal of the complete article.

OCULAR MOVEMENTS. C. D. VERRIJP, Arch. Ophth. **4**:73 (July) 1930; On the Analysis of Ocular Movements by C. D. Verrijp, communicated by Prof. J. Van Der Hoeve on Feb. 25, 1928, before the Royal Academy of Science in Amsterdam, and On a Further Analysis of the Ocular Movements by the same author, also presented before the Royal Academy of Science in Amsterdam in 1930.

These three articles are a mathematical study of the oculomotor movements. They form an original piece of experimental work dealing with certain questions of kinematics, largely a matter of the position of the center of ocular rotation. The article which appeared in the *Archives of Ophthalmology* is practically covered by the two more extensive and earlier articles presented before the Royal Academy of Science in Amsterdam.

In this paper, which will be considered first, the ocular rotations are likened to the motions that a rectangle could make in moving from one position to a second if one knew only these two positions. Drawings are presented to explain this. The same thing is then considered in an attempt to locate the eye in a series of successive positions without making a previous assumption about a center of rotation. An important and relevant assumption, however, is made; i. e., that

without sensible deviations the eye may make movements in which all of its points describe paths parallel to a flat plane. A most valuable conclusion from the work, as stated by the author, follows: "So one must admit that, at least in these series of experiments, the center of rotation was an ever changing one, and that only practically might one speak of one center of rotation, the coordinates of this center being the averages of the coordinates of the instantaneous centers." He calls further attention to important obligations to this center of rotation in ophthalmology so far as it plays an important part in the localization by x-rays of intra-ocular foreign bodies, and to the modification that retinal images may undergo as they appear through the various positions in lenses worn for ametropia.

The second and third of the articles mentioned contain a consideration of the place occupied by the eye in a number of successive positions and the formulas that were derived, enabling one to compute the location of the instantaneous center of rotation. As the author states in the first of these two articles, for convenience, these formulas were derived on the assumption that for each position a line, fixed in relation to the eye, and one fixed point on the line could be located. For the fixed line the visual line was taken (the line of sight of Gullstrand); for the fixed point, provisionally, the intersection of this line and the anterior surface of the cornea was taken. Only those movements are considered in which all points describe paths parallel to a horizontal plane. In deriving the curve of the instantaneous center of rotation, it is not necessary to determine at every turn the location of the fixed line. The knowledge of its direction is sufficient. So far as the fixed point is concerned, it is only necessary that this point be one with the eye, kinematically speaking. The location of the point, of course, must be known. As described in the American article, the experimental work herein detailed considered these facts: 1. If of two fixed points the place is known for a number of successive positions, one can determine how the eye has shifted, i. e., rotated. 2. Precision depends on the accuracy of the observations, and also on the minuteness of the displacements about which the observations are made

The third article is an explanation of the mathematics of these methods and their experimental realization.

In the conclusions of these articles Dr. Verrijp states: 1. Actually there is not only one center of rotation. 2. There is a scattered location of instantaneous centers of rotation which are mathematically connected. 3. These points are mathematically connected. 4. There is "practically" one center of rotation if one does not forget that the real motion is a much more irregular one. The author states that further investigations may disclose whether the amount of these deviations is different in different persons or is different in one person under different circumstances. Further investigations may also elucidate the cause of the deviations.

The articles, as already stated, are a deep scientific study, involving considerable mathematical knowledge, and are the results of a tremendous amount of laboratory work. If for no other reason than the conclusions stated, they are a momentous contribution to ophthalmologic physiology.

SPAETH, Philadelphia.

THE EVOLUTION OF THE SYNDROME OF THE LAST FOUR CRANIAL NERVES. F. J. COLLET, Rev. d'oto-neuro-opht. **8**:670 (Nov.) 1930.

The objective signs of the syndrome of the last four cranial nerves are: asymmetry of the soft palate, immobility of one vocal cord, hemiatrophy of the tongue with deviation of its point toward the diseased side and, especially, a displacement of the posterior pharyngeal wall toward the healthy side caused by a drawing over of the inert half of the pharynx by contraction of the healthy side. This displacement is apparently increased by the drawing over of the pillars of the palate of the sound side in the opposite direction. The chief subjective symptoms are dysphonia and difficulty in swallowing solids. The syndrome is caused by a lesion, not more than 1 cm. in diameter, in front of the occipital condyle, involving the contents of the jugular foramen and the adjacent anterior

condyloid foramen. Tachycardia and drooping of the shoulder, due to involvement of the tenth and eleventh nerves, are sometimes present.

Even in the case of traumatic lesions there is a certain chronology in the development of the symptoms: first, difficulty in swallowing and deviation of the tongue, followed, after some days, by hoarseness.

Three cases are reported, all due to neoplasms. The first case was due to metastasis from a tumor of the breast. The sequence of symptoms was: severe pain in the left retro-auricular region, inability to swallow solids, progressive hoarseness, pain in the left side of the neck, emaciation and paralysis of the left abducens nerve. Death, which occurred four months later, was preceded by an increase of the tachycardia, which was present at the first examination. There were: left lingual hemiatrophy, deviation of the point of the tongue to the left and drawing to the left of the uvula. The palate did not approach the posterior pharyngeal wall during phonation, and the left vocal cord was paralyzed. On phonation, the posterior pharyngeal wall was drawn to the right, and the right posterior pillar was drawn to the median line. The pulse rate was 128 per minute. The left side of the throat was anesthetic.

In the second case, the neoplasm appeared first in the right carotid region and then invaded the epipharynx. The symptoms were: otalgia on the right, some vertigo, but with good hearing, and painless difficulty in swallowing. On examination, there were: paralysis of the external rectus muscle, of the right vocal cord and of the right half of the palate; during gagging the posterior wall of the pharnyx was drawn toward the left, while the left posterior pillar was carried to the median line; the tongue was deviated to the right; the pulse rate was 120 per minute. At autopsy it was found that the tumor had not involved the interior of the skull; the point of the petrous bone was enlarged, which accounted for the paralysis of the abducens nerve; the tumor occupied the entire nasal and part of the buccal pharynx.

The third case was characterized by lingual hemiatrophy, pains in the head and neck, torticollis, palatopharyngeal hemiplegia and, later, partial paralysis of the common motor oculi and of the spinal accessory. The patient's mother had died of laryngeal tuberculosis, and in childhood the patient had suffered with an abscess of the ankle, which had been kept in plaster for several months. A tumor was found in the posterior pharyngeal wall. Some months later, intense headache, vomiting, diplopia, ptosis of the left lid, anisocoria and drooping of the left shoulder appeared. Lumbar puncture revealed a bloody fluid; the Bordet-Wassermann test gave negative results. A roentgenogram showed a cloud around the occipital foramen and the sella turcica. The clinical diagnosis was neoplasm of the cranial base, probably a sarcoma. The absence of laryngeal paralysis is accounted for on the supposition that the pressure on the pneumogastric nerve was high up and did not involve the laryngeal fibers. The process was not thought to be tuberculous. Antisyphilitic treatment was without benefit.

DENNIS, Colorado Springs, Colo.

REMARKS ON SOME FORMS OF SPASMS IN CHILDHOOD: THEIR CAUSE AND TREATMENT. A. DINGWALL FORDYCE, Brit. M. J. **1**:165 (Jan. 31) 1931.

The author reviews the various causes of neuromuscular spasms. They may be produced by local peripheral disturbances or may result from a primary lesion or abnormality of the central nervous system. He stresses the fact that it is not possible to dissociate the mental functions and the functions of nervous control in the nervous system. Spasms may occur from disturbances of the mental functions, without demonstrable abnormality in either the local neuromuscular physiology or the central seat of nervous control. Although the symptom of spasms might be considered under four headings, namely, those of congenital origin, those the result of gross intracranial abnormality, as a symptom of disease resulting from deficiency in vitamin A in infancy and as a symptom dependent on nervous hyperexcitability, congenital or acquired, temporary

or permanent; it is only the last group that the author considers in this paper. The significance of Chvostek's sign is considered. The author does not agree with those who disregard its significance in children of school age. In a series of 400 children examined in a consecutive series, 7 per cent showed signs of facial irritability. In a child of unstable nervous constitution, this appears to depend on the degree of alteration in the systolic blood pressure, brought about by the administration of a particular internal secretion. In the cases of asthma in which the patients were treated with ephedrine and in which there were symptoms of collapse, lethargy and a tendency to faint shortly after taking the medicine, facial irritability was marked in five cases, while only in two, in which facial irritability was negative, did symptoms of a minor degree occur.

The significance of the calcium content of the blood in spasmodic conditions is next considered. The author concludes that, apart from infantile spasmophilia, there does not appear to be a definite relationship between the calcium content of the blood and spasmodic conditions or a positive facial irritability.

The relationship of the endocrine system to spasms is briefly reviewed. It would seem that the suprarenals and the thyroid tend to produce sympathicotonia, and thus the administration of the products of these glands of internal secretion is successful in the treatment for vagotonia and certain anaphylactic reactions, notably asthma and certain conditions of the skin that seem to occur frequently in this type of person.

The importance of emotion and instinct in the production of spasmodic conditions in childhood is emphasized. Chorea is considered to be a symptom and not a disease. It may be due to a multiplicity of causes. Two approaches in treatment are evaluated, namely, the one directed to the alteration of the calcium metabolism, and the other to control of the nervous system with phenyl-ethyl-hydantoin. The author concludes that if severe and acute chorea occurs with abnormal amounts of calcium in the blood, the possibility that lack of calcium is an essential element in the development of the symptom is excluded. Treatment with phenyl-ethyl-hydantoin is considered somewhat dangerous because of a poisoning due to its use called "Nirvanol sickness."

Convulsions and fits are next considered. The various causes are briefly reviewed, and the treatment for each respective group is briefly outlined.

FERGUSON, Niagara Falls, N. Y.

OXYCEPHALY. G. WORMS and R. CARILLON, Rev. d'oto-neuro-opht. **8**:736 (Nov.) 1930.

Roentgenography is of prime importance in the diagnosis of oxycephaly, and exposures in the anteroposterior, lateral and vertical (base of the skull) planes should be studied. The deformity does not always involve the cranial dome; it may be located at the base and cause a basilar lordosis. Other cases show a marked asymmetry of the face—as though it were twisted to one side—which results in marked deviation of the nasal septum and reduction in the size of the orbit, the middle cerebral fossa and the petromastoidal mass of the side to which the face is twisted.

The classic conception that precocious closure of the coronal and sagittal sutures, by thus increasing intracranial tension, is one of the essential causal factors does not explain the dystrophies of the facial mass nor the hyperostoses or exostoses that may be seen in the cranial walls. Furthermore, closure of the sutures is not a constant observation, nor is optic neuritis or old papillary edema found in certain cases. In oxycephaly the frequency of various malformations, such as harelip, bifid uvula, syndactylia or polydactylia, congenital cardiac lesions and umbilical hernia would suggest the probability that some trophic disturbance of the cranial skeleton is the cause. Owing to an unusual malleability from defective ossification, the cranial box may be distended by the normal pressure of its contents or by increased pressure due to intercurrent hypertensive reactions. The repeated nasopharyngeal infections in the young person offer an infectious

focus which might cause this reaction. Endocrine disturbances and alcoholism in the progenitors have often been noted in the history of patients with oxycephaly.

Exophthalmos is one leg of the tripod supporting the craniofacial dysostosis of Crouzon, and it has been found in oxycephaly. It is due to orbital distortions. The nystagmus that sometimes occurs is to be regarded as due rather to errors of refraction (usually astigmatic hypermetropia) than to labyrinthine disturbance. Optic atrophy is a grave complication. Nordmann thought that the atrophy is from hypertension and not from neuritis, but in oxycephaly there is gray atrophy, and, as compression usually causes white atrophy, the former cannot be explained by it. Direct pressure on the optic nerve does not cause papillary edema, and narrowing of the optic foramen as a cause of postedematous atrophy is not accepted. However, in one of the cases reported, there was a unilateral narrowing of the foramen on the side of the more affected eye (the right). It was oval, with its greatest diameter vertical. The right papilla was smaller than the left, but appeared to be normal. The visual disturbances consisted in a marked reduction in the fields, hemeralopia and changes in the chromatic sense.

In no case were labyrinthine disturbances found, aside from a mild hypoexcitability. Three cases showed cochlear hearing defects, and when they were unilateral they were on the same side as the ocular disturbances. Two cases are reported in detail and are accompanied by roentgenographic reproductions.

These observations tend to show that there are transitions between pure oxycephaly and hereditary craniofacial dysostosis and that the two are closely related.

DENNIS, Colorado Springs, Colo.

TRAUMATIC CORTICAL PARAPLEGIA. A. LÉVI-VALENSI and EZES, Encéphale **25**:667 (Nov.) 1930.

Paraplegia, as a result of arterial changes, tumors and traumatisms, is by no means uncommon, but as a cortical phenomenon in the adult it is extremely rare. A youth, aged 16, was struck on the vertex in February, 1928. Hemorrhage was profuse; complete unconsciousness supervened for forty-eight hours and was followed by a gradual restoration of mental function. The lower extremities were paralyzed in extension, the left arm in hyperflexion. Sensation was undisturbed, except that the sensation of pain was abolished in the left arm. In May, 1928, neurologic examination revealed moderate atrophy of the lower extremities, and paraplegia in extension, with the right foot in a varus equinus position and minimal contracture. The loss of motion was considerable, and the patient was unable to walk or support himself unaided. The ability to extend the feet on the legs was lost, while flexion was fairly well preserved; there was some preservation of flexion of the leg on the thigh, which was better on the right. Abduction of the thigh was preserved; adduction was diminished. Flexion and extension of the thigh on the abdomen was lessened, more so on the left. The left upper extremity manifested atrophy, especially of the deltoid and hand. All movements were possible but weakened in force, and sensation in all modalities was absolutely normal (the patient showed good intelligence and cooperation). The reflexes were increased; the cutaneous abdominal reflexes were normal; the plantar responses were of the extensor type. There was pronounced vasomotor alteration (sweating) of the affected extremities. The electrical reactions were normal. Spinal puncture yielded negative results.

In view of the marked cranial depression, an operation was performed on May 24, and was followed by death. Autopsy revealed marked adherence of the meninges over the middle portion of the brain, overlying a depression, 5 or 6 cm. in diameter, extending to both hemispheres but more marked on the right. The right paracentral lobule was destroyed in its anterior three-fourths, and the lesion extended into the convolution of the corpus callosum. On section, it was seen that a small subcortical lesion had occurred in the centrum ovale, and was connected by a sort of peduncle to the cortical lesion.

According to Benisty, such cases fall into three types: partial peripheral paralysis, asynergic or incoordinated paralysis and a spasm or contracture type. The case reported would perhaps fit best into the first group. The lack of continued sensory disturbance seems to cast doubt on the specific sensory function of the posterior portion of the paracentral lobule. The diplopia with strabismus is to be imputed probably to inhibition of the cortical centers for the ocular musculature. The atrophy was probably more than could be accounted for by the central lesions, per se. The cord was not examined, but it seems improbable that such a uniform pathologic process as was shown could be the result of direct trauma. It may be possible that there was a sort of "echoing or reverberation" of a cerebral lesion to the anterior horn cells of the cord.

ANDERSON, Los Angeles.

SOME PRINCIPLES IN THE TREATMENT OF BEHAVIOR PROBLEMS IN CHILDREN. LAWSON G. LOWREY, J. Nerv. & Ment. Dis. **73**:62 (Jan.) 1931.

Measures applied in the treatment for behavior problems in children have a double task: (1) to attempt to care for present behavior problems; (2) prevent the development of more serious problems in the future. Treatment may be divided into a direct and an indirect form. The first includes the physical and psychotherapeutic treatment of the child himself. The second connotes the social manipulative measures intended to make the environment of the child into a source of positive instead of negative stimulation. The child himself must be regarded as a complicated personality at any stage, living in a variety of groups, each presenting dynamic features that stimulate behavior either positively or negatively. Positive stimulation is defined as that which makes for a wholesome personality, while negative stimulation produces disorganization of the personality. Manipulation of the schooling so that the child receives adequate placement in the light of his abilities, with a consequent feeling of achievement, is important in developing recognition of him as an individual. Treatment of the affectional relationships of the child in the family, its jealousies and the feeling of deprivation, demands a more subtle understanding. Changing the child's environment, companionship and play brings him under a different type of influence. Often intensive psychotherapy of the mother or other important individuals in the family becomes necessary.

Five groups of factors in the causation of behavior disorders have been worked out. Group 1 includes the gross mental deviations, psychoses, feeblemindedness, epilepsies and the postencephalitic behavior disorders. Group 2 is composed of gross physical deviations. Group 3 consists of the neuroses, and demands in the child as in the adult intensive and special therapy to stabilize the personality. These three groups demand expert training and individual attention if anything is to be accomplished. Group 4 contains the complexes and conflicts of less than the neurotic level and lies intermediate to groups 3 and 5. Group 5, presumably the simplest group, represents complexes and conflicts brought about through ignorance and faulty methods of training; it involves much parental education. The ignorance of the parents is not always based on an intellectual difficulty but is often due to their emotional conflicts. In groups 4 and 5 lie the battle-grounds of the psychologist, the sociologist, the psychiatrist and the educator, and there are many different kinds of clinics representing this overlapping of technic. No one group has all the answers to the problems of human behavior.

It is best to begin with the simplest and most direct measures in affecting the situation, leaving the more complicated emotional tangles to be dealt with later as seems important. It is wise to proceed slowly in the treatment of these emotional tangles so that confidential rapport with the child is permitted to develop, and this may be said to be the most important point involved in the problem that is handled successfully.

HART, Greenwich, Conn.

DILATATION OF THE VENTRICLE OF THE SEPTUM PELLUCIDUM. E. MEYER, Arch. f. Psychiat. **91**:9, 1930.

The author reports five cases in which postmortem examinations have shown dilatation of the ventricle of the septum pellucidum.

In case 1, during the course of the disease, symptoms pointing to a possible metastasis in the brain developed in a patient who was suffering with what appeared to be a mediastinal tumor. The picture was characterized by epileptiform convulsions, left-sided paralysis, choked disks and, toward the end, a state of delirium. Encephalography showed dilatation of the ventricles and suggested the presence of a tumor. Postmortem examination showed that the whole ventricular system was dilated, with a special accentuation of this in the ventricle of the septum pellucidum. No tumor was found. In fact, outside of some thickening of the meninges and fresh hemorrhages in the corpus callosum, no pathologic condition was found.

In case 2, a more or less similar condition is described, but without any definite indications of disease outside the central nervous system. Here, too, an enlargement of the ventricle of the septum pellucidum was the only important discovery at postmortem examination.

In case 3, the history of ten years' duration of psychic deterioration and a picture of dementia without any neurologic symptoms were the only manifestations. Postmortem examination revealed an enlargement of all the ventricles, but especially of the septum pellucidum. No other pathologic changes were discovered.

In case 4, the clinical picture started with mental symptoms about a month before death. The patient complained of paresthesia and hallucinations. She became delirious, and toward the end the knee reflexes were absent. In this instance, also, a marked enlargement of the ventricle of the septum pellucidum was the only outstanding pathologic change found at autopsy.

In case 5, a schizophrenia-like picture developed about four years before admission. Toward the end, the patient showed recurring attacks of confusion and delirium. At autopsy in this case, too, the only feature of importance was a dilated ventricle of the septum pellucidum.

In reviewing the literature, the author finds that this condition is rather infrequent, but that it may be found in cases of different origin. It is found in cerebral arteriosclerosis and in some tumors of the brain, especially in those that are localized in the cerebullum, in the pons or under the lateral or third ventricles. Exactly what the mechanics of such a condition are and why they should occur in some cases in connection with other diseases and in other cases apparently without any other pathologic condition remains at the present unknown. Meyer points out that normally the septum pellucidum should consist of the two walls with either no space between them or with only a potential space.

MALAMUD, Iowa City.

PARINAUD'S SYNDROME WITH LABYRINTHINE DISTURBANCES: THE PHYSIOPATHOLOGIC MECHANISM OF THIS SYNDROME. A. KREINDLER and A. SCHEIM, Rev. d'oto-neuro-opht. **8**:731 (Nov.) 1930.

This observation concerns a woman, aged 59, who, following an ictus, had a right hemiplegia with facial paralysis of the left side, paralysis of movements of the eyes upward, deviation of the right eye downward and outward, mydriasis in the right eye and spontaneous nystagmus. At the first examination, the urine contained no albumin or sugar, but later the sugar content was 3.6 per cent. Improvement soon began, and, as these clinical signs receded, cerebellar signs appeared: dysmetria, a tendency to fall backward and to the right and adiadokokinesis on the right.

Attention is called especially to the labyrinthine disturbances: spontaneous nystagmus, which disappeared after ten days, hyperexcitability to the galvanic current and hypo-excitability to the caloric test. They frequently occur in cases of associated paralysis of the eyes (Duverger and Barré, Léri and Bollak and

Oljenik). Babinski, Vincent and Barré and Magnus regarded deviation of the eye downward and outward as a labyrinthine symptom. After extirpation of one labyrinth in a monkey, Bartels and Magnus observed a vertical deviation upward of the globe. Labyrinthine disturbances are not caused by the paralysis but by reflex spasms of the muscles, originating in the labyrinth. Magnus and de Kleyn have shown that the distribution of tonus in the ocular muscles is regulated by tonic labyrinthine and neck reflexes, especially in animals with panoramic vision, but they also exist in man. The labyrinthine reflexes are exercised especially on the superior and inferior recti and the two oblique muscles; the neck reflexes, on the external and internal recti. These facts are useful in the interpretation of the physiopathologic mechanism of disturbances of associated movements of the eyes and the rôle that the labyrinth plays therein. In man, voluntary innervation of the ocular muscles is by way of the lower motor mechanisms, which are phylogenetically older and are in the nature of tonic labyrinthine and neck reflexes. In man, the tonic reflexes of the eyes are dissociated in the sense that functional association of the ocular muscles (vertical and lateral gaze) is made in the voluntary mechanism, independently of the position of the head in space. In cases of Parinaud's syndrome, the lesion affects the oculolabyrinthine association pathways. The mechanism of the tonic labyrinthine reflexes on the eyes is altered, and the voluntary innervation, coming from above, can no longer use this pathway to carry the eyes upward and downward. The alteration of the tonic reflexes in the neck on the eyes could, by analogy, be invoked in the pathogenic interpretation of Foville's syndrome.

DENNIS, Colorado Springs, Colo.

THE CONSTITUTIONAL VARIATIONS OF THE PSYCHOMOTOR FUNCTIONS AND THEIR RELATIONSHIP TO PHYSIQUE AND CHARACTER. M. GUREWITSCH and N. OSERETZKY, Arch. f. Psychiat. **91**:286, 1930.

This investigation of the functions of the psychomotor apparatus and the relationship that they bear to the Kretschmer types was carried out on a large number of normal persons. Altogether, over 8,000 persons were examined, but the results were computed on the basis of the observations in about 5,000 of these because the others did not fit into definite types. The group consisted of men and women, ranging in age from 16 to 50 years, and representative of the following five physical types: leptosome, athletic, pyknotic, infantile and dysplastic. The functions studied were: (*a*) frontal, the ability to perform simultaneous movements and special motor formulas; (*b*) fronto-extrapyramidal, the rate of movement and the speed with which adjustments to new movements could be made; (*c*) pyramidal, the power and the precision with which movements were made; (*d*) extrapyramidal, rhythmicity and automatic movements; (*e*) corticocerebellar, static and dynamic coordination.

In addition to the division into the physical types described, the material was also divided into types of character: schizothymic, cyclothymic, epithymic and labile. The underlying purpose was to determine whether the functions of the psychomotor apparatus correspond to any of the types of physical or mental make-up. The results were:

Cyclothymic-pyknotic persons showed well developed extrapyramidal, corticocerebellar and fronto-extrapyramidal functions. The frontal and pyramidal components were only mediocre and there was a definitely low value for manual dexterity (special formulas). Persons of the schizothymic-leptosome type scored a high rating in the frontal functions, especially in manual dexterity; the corticocerebellar and pyramidal components were mediocre, whereas the extrapyramidal and frontal extrapyramidal components were poor. Persons of the epithymic-athletic type scored a high rating in the pyramidal functions; the frontal and fronto-extrapyramidal components were below medium; the same was true of the extrapyramidal components and the manual dexterity, whereas the corticocerebellar functions were somewhat above medium. Persons of the labile-infantile type

showed a fairly high development of the extrapyramidal and corticocerebellar functions, whereas the frontal and pyramidal components were low.

When the physical and mental types did not correspond, that is, when, for instance, a pyknotic person showed a schizothymic make-up, etc., these functions became mixed in such a way that the functions in which the pyknotic person usually excelled were somewhat lower, but this was compensated for by an improvement in those that were carried out particularly well by the leptosome type. It was also observed that, generally, the psychomotor functions were more closely related to types of character than they were to types of physique.

MALAMUD, Iowa City.

FACIAL NEURALGIA TREATED BY RETROGASSERIAN NEUROTOMY. AMZA JIANO, D. PAULIAN AND D. TURNESCO, Rev. d'oto-neuro-opht. **8**:614 (Oct.) 1930.

A married woman, aged 56, entered the hospital with facial neuralgia on the right side. The past history was not significant. The pain, which had been present for six years, followed dental caries. At first the pain was localized in the right upper gum and was amenable to treatment with anodynes, but later it extended to the entire right half of the face. In spite of a variety of treatments, including two injections of alcohol, relief was only temporary. A resection of the sensory root of the trigeminus was done under local anesthesia, following a tarsorrhaphy. On exposure of the ganglion, a sharp hemorrhage occurred. The sensory root was severed, but the motor root was not identified. On account of the bleeding, a strip of gauze was packed in the depth of the wound and was led out through the lower angle of the incision, which was closed with catgut. The operation lasted three hours. The postoperative course was uneventful except for a rise of temperature to 39 C. (102.2 F.) on the second day and again in the period between the tenth and the sixteenth days. The pain was immediately relieved.

An examination of the patient two months later revealed a slight facial asymmetery but no paralysis; sensation was abolished in the domain supplied by the fifth nerve on the right; the corneal reflex was absent and the cornea was intact. The right masseter muscle was not paralyzed.

Injections of alcohol have a practical importance in the diagnosis of facial neuralgia and in the prognosis of pregasserian neurotomy. When the injections do not cause even a temporary amelioration of the pain, the condition is not one of essential facial neuralgia, and neurotomy will be unsuccessful.

Of the three surgical methods of treatment, section of the branches, removal of the ganglion and section of the sensory root, the last is preferable. It can be made at one of two points: in the neighborhood of the bulb, or just before its termination in the ganglion. Lesions of the masseter nerves and trophic disturbances of the eye from traumatism of the ganglion are dangers of the operation. Dividing the root near the bulb, in accordance with Dandy's proposal, has the advantages of greater visibility and the possibility of a partial division of the root. According to Dandy, division of three fourths of the root is sufficient to relieve the pain and the disagreeable sensation in the areas supplied by the nerves, and trophic troubles do not occur. On the other hand, this method of operation is much graver than the other methods mentioned, and there is danger of injuring the eighth nerve. The importance of severing the masseter nerve is not great because, owing to the anesthesia in the mucosa, these patients will chew on the healthy side. Keratitis can be partially avoided by a tarsorrhaphy preliminary to the operation. Relapses are possible after partial resection and even after total division, but they are exceptional.

DENNIS, Colorado Springs, Colo.

A CONTRIBUTION TO THE STUDY OF HYSTERICAL APHASIA. GIOVANNI DI LALLA, Ann. di nevrolog. **44**:81 (May-June) 1930.

Hysterical disturbances in speech may be as numerous as those caused by organic lesions. The most common forms include simple aphonia, mutism,

anarthria and dysarthria. True aphasic disturbances in hysteria are rare. According to some observers, all types of organic aphasia may be observed in hysteria. Cases of word blindness, word deafness, "aphemia" and agraphia have been reported.

The author describes a case of motor aphasia in a woman, aged 28. In addition to the aphasia, she presented typical hysterical right hemiplegia, right hemianesthesia and right homonymous hemianopia. All of these symptoms, including the aphasia, disappeared after the application of an electric current. The difficulty in speech had existed for one year, and had set in following a convulsive attack. At the onset, the vocabulary of the patient was reduced to a few intimate words, such as her name, the name of her husband, etc. To other questions she would answer: "I cannot." At other examinations, she had difficulty in pronouncing words that she had previously spoken, but pronounced new words easily. She did not show disturbances in writing. She understood everything that was said to her, carried out simple as well as complicated orders well, and could read aloud.

Hysterical aphasia is distinguished from organic aphasia by the following: 1. There is an absence of agraphia in the former condition. 2. There is an irregularity in the aphasia — the person with organic aphasia pronounces only a few, and always the same words, while the person with hysterical aphasia has a richer vocabulary, pronounces new words, and often forgets words that had previously been pronounced. 3. The person with organic aphasia uses oligosyllabic words, and usually those that are emotionally toned (curse words, etc.), while the person with hysterical aphasia has no difficulty with polysyllabic words and those of intellectual content. 4. While the former tries to answer all questions with his few words and becomes impatient with his difficulty, the latter sits quietly and says, "I cannot." 5. The intelligence, at least in some particular sphere, is lowered in the person with organic aphasia; it is not in the hysterical person. 6. The person with hysterical aphasia has more difficulties in speech while she is being examined than when she is talking with other patients. 7. Sudden recovery following electrical stimulation speaks for hysteria.

IMPASTATO, New York.

THE PATHOLOGIC NEUROPHYSIOLOGY OF A PUPPY FOLLOWING THE REMOVAL OF A HEMISPHERE. B. L. KLOSSOVSKY, Collected Papers, The Brain Institute, Leningrad, Bechterew Memorial Volume **3**:326, 1929.

In newly born puppies one hemisphere was removed by making a large opening in the temporal bone; in this way no hernia cerebri formed afterward. The empty space left by the removal of the hemisphere was quickly occupied by the compensatory growth of the remaining hemisphere. In the operation only the neopallium was removed. There was a marked retardation in the growth of the puppies for forty or fifty days as compared with the growth of the control puppies which were not operated on. Immediately after the operation, the puppies lost about 20 Gm. daily and this sometimes increased to 40 Gm. during the subsequent weeks. Six weeks after the operation, the dogs reached their preoperative weight. Some functions, however, developed more rapidly in the experimental animals than they did in the controls. Thus, while the latter still crawled on their abdomens the former ran quickly and accurately. Lapping of milk appeared also much earlier in the experimental animals than it did in the controls. Between the ages of 40 and 50 days the following phenomena appeared: the puppies suddenly began to limp on the side contralateral to the operation; tonus on that side was also increased. These phenomena persisted for varying periods of time, lasting from several hours to several months. The histogenetic data show that this age corresponds to the period of intensive myelinization of the brain, especially of the pyramidal tracts and the motor areas. The sooner the hemisphere is removed after birth, the less pronounced are these phenomena.

When a hemisphere is removed in an adult dog one can notice that the dog sways toward the side of the removed hemisphere while he runs. There is also marked hemianopia; the whole world appears to the dog to exist on one side only. In dogs in which the hemisphere has been removed at birth, the behavior does not differ from that of control dogs after the age of 9 months. However, after this period the dogs begin to exhibit behavior that is strikingly similar to that which one finds in dogs in which the hemisphere is removed in adult life.

The author postulates that the phylogenetic functions of the subcortical centers are suppressed by the remaining hemisphere. This leads to the conclusion that the dog with both hemispheres removed is a more efficient animal than the one in which one hemisphere was removed at birth or afterward.

KASANIN, Boston.

FETAL ADENOMA OF THE HYPOPHYSIS AND DERMOID CYST OF THE HYPOTHALAMUS. PHILLIP F. SHAPIRO, Arch. Path. **11**:22 (Jan.) 1931.

The hypophysis and the adjacent tissues are the site of a great variety of tumors. The hypophysis itself is composed of several elements, and each is the potential anlage of a separate type of neoplasm. The pars nervosa gives rise to gliomas and the pars glandularis to simple hyperplasias, adenomas and carcinomas of many kinds. Even the dubious pars intermedia has been credited with certain tumors. Each of the cellular elements in the pars anterior has its special prototype in neoplasms. Thus, hypophyseal tumors have been described that were composed entirely or chiefly of eosinophil cells, basophil cells, principal cells, "transitional cells" (Kraus), pregnancy cells and rarely even fetal indifferent cells. All possible combinations of these cells may be seen in the mixed adenomas, and in a single hypophysis two or more varieties of these tumors may be found separately. The relative frequency of the various types of tumors is roughly proportional to the relative numbers of the type cells. Thus, eosinophil adenomas are the most common, fetal cell adenomas are rare and pars intermedia tumors practically never occur in man.

A fetal cell adenoma of the hypophysis and a dermoid cyst of the hypothalamus have been reported. The first tumor resembled one of the so-called "pars intermedia tumors." It originated, however, not from a pars intermedia, but from the midportion of the pars anterior. It was composed not of the specific differentiated pars intermedia cells, but of undifferentiated fetal cells. If man has a pars intermedia comparable with that of lower animals, it is rudimentary, variable and functionless. There is, as yet, no histologic proof establishing its presence.

The dermoid cyst belonged to the group of inclusion tumors of the hypophyseal area. It was derived from embryonal rests of cranial external ectoderm included during the invagination of the brain vesicles. It was related to Erdheim's tumors, derived from rests of oral ectoderm invaginated with Rathke's hypophyseal pouch. It involved the hypothalamus, leaving the hypophysis relatively intact, and yet produced the diabetes insipidus and the diabetes mellitus usually ascribed to hypophyseal lesions. The diencephalon rather than the hypophysis is chiefly responsible for these syndromes.

WINKELMAN, Philadelphia.

TRANSVERSE MYELITIS WITH APOPLECTIFORM ONSET: A CLINICAL AND PATHOLOGIC CONTRIBUTION TO THE STUDY OF MYELOMALACIA. L. CONDORELLI, Riv. di neurol. **3**:233 (July) 1930.

The author describes a case of transverse myelitis which developed suddenly twelve days after an attack of influenza. The tetraplegia was not preceded or accompanied by an increase in temperature. Two days after the ictus, some movements reappeared in the upper extremities. The deep reflexes of the upper extremities were very sluggish, while those of the lower extremities were absent. There was a loss of sensibilities from the third intercostal space downward. The patient died a few days later from bronchopneumonia.

Macroscopically, the spinal cord appeared soft at the level of the eighth cervical and first dorsal segments. Histologically, at this level there was a complete necrosis of the tissue. The area of softening extended to the third dorsal segment. In the sixth dorsal segment there was a small hemorrhage in the anterior portion of the posterior column; in the ninth dorsal segment, an area of softening in the left anterior horn; in the sixth cervical segment, a small area of softening in the anterior columns. The lesions all were of a degenerative type; no inflammatory manifestations were found. The most important lesion for an explanation of the symptomatology of a transverse section of the spinal cord was the ischemic softening at the level of the eighth cervical and first dorsal segments.

The author emphasizes the fact that there were no lesions involving the blood vessels of the pia at the level of the eighth cervical and first dorsal segments and that the anterior spinal artery appeared normal. However, hyaline thrombi were found in the intramedullary blood vessels at the level of the eighth cervical segment. The absence of inflammatory lesions makes the thrombotic process a purely toxic one. The vascular origin of the lesions is proved also by the fact that the most involved territories were those of the anterior medial arteries, the anterior radicular arteries and the arteries of the anterior columns. The author believes that the medial anterior artery underwent first a process of thrombosis, followed immediately by hyaline thrombi in the other vascular territories. The author objects to the term myelitis in cases of this nature and prefers the term myelomalacia used by Schmidt.

FERRARO, New York.

RESPIRATORY SEQUELAE IN A CASE WITH THE SYNDROME OF THE POSTERIOR LACERATED FORAMEN, FIFTEEN YEARS AFTER THE TRAUMATISM. GIORGIO FERRÉRI, Rev. d'oto-neuro-opht. **8**:665 (Nov.) 1930.

Ferréri bases this article on the study of a patient who fifteen years previously had received a pistol-shot wound; the bullet was still in situ in the left jugular foramen. The patient had paralysis of the left half of the palate, the superior constrictor muscle and the larynx, and a slight hypotrophy of the left trapezius muscle. Tracings were made of the movements of the larynx, the thorax and the abdomen both during tranquil breathing and during phonation. During tranquil breathing the larynx did not show movement; the movements of the left side of the thorax were, in general, better than those of the right; abdominal respiration was greater than the thoracic, and the movements were better on the right side. "There is almost a compensation between the thorax and the abdomen." During phonation, the larynx showed irregular oscillations; the patient began to speak only at the end of expiration, thus losing a large quantity of air; the left side of the thorax moved much less than the right; the abdominal movements showed shorter inspiratory curves on the left than on the right side. It is to be noted that, during tranquil respiration, the left side of the thorax was more active than the right, contrary to what one would expect and to what occurred in the abdominal movements; on the other hand, during phonation, movements of the right side of the thorax were more ample and regular than those of the left side. During the fifteen years that had elapsd since the injury, compensatory phenomena had been established so that the two types of respiration were clearly recognizable. During tranquil respiration, the respiratory movements of the side of the chest corresponding to the lesion (the left) were more ample but more irregular than those of the right side, which is in agreement with classic physiologic observations. On the contrary, during phonation the excursions of the left side were less ample and more irregular, and the expiratory movements were interrupted by rapid and superficial inspiratory movements, showing an insufficient and abnormal respiratory mechanism.

The article is illustrated with unusually good reproductions of roentgenograms.

DENNIS, Colorado Springs, Colo.

THE INFLUENCE OF THE GALVANIC CURRENT ON THE REGENERATION OF INJURED NERVES (EXPERIMENTAL INVESTIGATION). I. A. PIONTKOVSKY, Arch. f. Psychiat. **91**:269, 1930.

Although it is generally known that the galvanic current exercises a beneficial effect on the healing process of severed nerves in that it helps to promote regeneration of the tissue, actual experimental investigations are not numerous. Piontkovsky reports the results of investigations carried out on the sciatic nerves in rabbits. The nerves were cut under local anesthesia on both sides and then the nerve ends were straightened out and the wound was closed. One side was subjected to the effects of a galvanic current of from 2 to 6 ma., beginning with exposures for fifteen minutes and ending with exposures lasting forty-five minutes. The other side was allowed to regenerate without the effect of the electrical current. This was carried out for different lengths of time (from seven days to two months) following which the animals were killed and the nerves examined histologically. The clinical and histologic observations led the author to the following conclusions:

The galvanic current exerts a pronounced influence on the function of the paralyzed limb after the operation. Under this influence there was improvement in movement of the treated extremity as compared with the one that was not treated. The treatment also had a good healing effect on the trophic ulcers in that they were not as large or as deep on the treated side as they were on the other.

The current has a definite influence also on the histologic picture. The microscopic picture of the regenerated nerve subjected to the galvanic current showed the same stage of progress at the end of the third week as was found in the untreated nerves two months after the operation. When the treatment was carried on for two months the sectioned nerve showed exactly the same picture as is found in the normal nerve. In the nerve stump that had been subjected to the influence of the galvanic current there was a much more pronounced growth of young axis cylinders than on the opposite side. MALAMUD, Iowa City.

A BRIEF PSYCHOLOGICAL ANALYSIS OF A PATIENT WITH ULCERATIVE COLITIS. CECIL D. MURRAY, J. Nerv. & Ment. Dis. **72**:617 (Dec.) 1930.

Murray analyzes the case of a woman, aged 37, who complained of sterility. The chief physical observation was an irritable sigmoid colon with an ulcerated bleeding mucosa. The patient had had from one to ten stools a day for the preceding three years. This condition had begun while she was traveling in Europe with a lover who was twelve years older than herself and who was married to a woman whom he had at first called his sister. The onset of the colitis coincided with the first occasion when they registered at a hotel as husband and wife. The mere mention of the first meeting with her lover gave the patient an attack of cramps. She recalled the funeral of her mother and how the latter had been borne away in a coffin. Subsequently, at the age of 6, while in a dark toilet, she had visions of her mother, and certain dreams of reincarnation suggested a wish to return to her mother. Whenever her stepmother threatened to beat her the patient used to have a desire to defecate. Between the ages of 16 and 19, she collected publishing material with an older woman named Ruth, aged 40, for whom she developed a very strong attachment. Her subsequent lover reminded her of Ruth because of his marriage, for Ruth had left the patient to get married. The author discovered an unconscious castration wish against the lover which developed after an abortion for which the latter was responsible. According to the author's interpretation the patient had an incestuous love for her father, which was expressed in her love for a married man, as a father substitute, who, like the father, was tied to another woman. The castration wish was interpreted as a desire to transfer the father into the mother substitute, like Ruth. Her libidinous fixation on the anal level is demonstrated by dreams of rubbish, severed heads of Negroes and taking refuge in toilets. The desire to

have a child is interpreted as a desire to cling to the lover as well as an indirect method of revenge against him for causing the abortion. Much of the patient's nervous tension is attributed to the conflict of being unmarried, while yet being bound to a married lover.

HART, Greenwich, Conn.

INDICATIONS AND CONTRAINDICATIONS OF ENCEPHALOGRAPHY AND VENTRICULOGRAPHY. EUGENE P. PENDERGRASS, J. A. M. A. **96**:408 (Feb. 7) 1931.

The indications and contraindications for ventriculography and encephalography are precise; each has a definite field in the diagnosis of lesions of the brain. Ventriculography is the procedure of choice in cases of increased intracranial pressure, including unlocalized tumor, abscess, basal arachnoiditis or any condition causing block or deformity of the ventricular system. In unlocalized cerebral lesions without increased pressure, it is contraindicated because of the mortality. The advantages of the procedure are the complete outlining of the ventricular system and the localization of lesions; the disadvantages are the necessity for two openings with the trephine, the possibility of hemorrhage from the puncture of a tumor, reaction following the introduction of air, the mortality rate, the possible failure to find and drain both ventricles and the fact that the subarachnoid space may not be delineated. Encephalography is indicated in cases in which the symptoms are obscure, as in those following trauma, inflammation and senility, epilepsy, hemorrhage, tumors, hemiplegias and injuries caused at birth or in any condition that would distort or obliterate the subarachnoid spinal fluid pathways. It is contraindicated in all cases having a pressure of 20 mm. of mercury or more in the horizontal position; for this reason, obstruction of the aqueduct or of the third or fourth ventricle should be ruled out. The advantages of this procedure are its simplicity, rare mortality, the visibility of the subarachnoid pathways, the diagnosis of lesions on the surface as well as of those within the brain, and of tumors without greatly increased pressure, the possibility of observation of lesions in patients under treatment for epilepsy and the relief of posttraumatic headache. The discomfort of the patient, the danger of foraminal hernia in the presence of tumor of the posterior fossa and the difficulty of securing outlines of the posterior and inferior horns of the ventricles constitute the disadvantages. Errors in surgical technic in either procedure render a correct interpretation of the films impossible.

JENKINS, Indianapolis.

PARKINSONISM DUE TO POISONING BY CARBON DISULPHIDE. F. NEGRO, Rev. neurol. **2**:518 (Nov.) 1930.

Parkinsonism is not a disease but a syndrome that may be produced by a variety of causes. The substantia nigra and the globus pallidus are involved to different degrees in different cases. Recently, a number of instances of parkinsonism following intoxication with carbon disulphide have been recognized. The patient whose case is reported was a worker in artificial silk who presented no indication of encephalitis or venereal disease. Two years after beginning work, general malaise, weakness — especially in the lower limbs — and impotence developed insidiously. Some months later, tremors began in the upper limbs, with psychic disturbances characterized by depression and irritability. At the time of examination, a clinical picture of parkinsonism was evident. The tremor was constant during rest, was interrupted by voluntary movement, and could be controlled momentarily by the patient. In walking, the patient held the arm flexed and close to the abdomen, with the fingers enclosing the thumb. Speech was slow and monotonous, and writing was typical of parkinsonism. There was a definite cog-wheel phenomenon, with an increase in reflexes, but no Babinski sign. The pupils reacted normally; there was diminution of convergence, but no nystagmus. No sensory disturbances were elicited, and the electrical reactions were normal. The serology was normal.

Aside from the interest in the etiology in this case of parkinsonism, the author emphasizes the importance of the cog-wheel phenomenon as indicating hypertonicity of the sarcoplasm, thereby differentiating an extrapyramidal disease from simple intoxication due to alcohol, mercury, tobacco, etc. The clinical symptoms of poisoning due to carbon disulphide are similar to those of poisoning due to carbon monoxide and manganese. Moreover, cyanides have occasionally produced similar symptoms. The occupational hazard is noted.

FREEMAN, Washington, D. C.

PERMANENT CONJUGATE DEVIATION OF THE EYES IN EPIDEMIC ENCEPHALITIS WITH CONTRALATERAL LOSS OF POSTURAL REFLEX AND MUSCULAR HYPOTONIA: A PECULIAR SYNDROME. L. TAUSSIG and T. DOSUŽKOV, Rev. d'oto-neuro-opht. **8**:679 (Nov.) 1930.

Two cases of epidemic encephalitis with a clinical picture different from the usual parkinsonism were observed. Both showed deviation of the eyes to the right, of a duration of two years in one case and of ten years in the other. Both showed spontaneous nystagmus: in the first, it was mixed, oblique upward and rotatory to the right; in the second, there was nystagmus to the right in the right eye and to the left in the left eye. Both showed: permanent deviation of the eyes upward and to the right, which persisted during sleep; narrowing of the right palpebral fissure; diminished muscular tonus, and a failure of the reflexes of posture on the left. In the second case, there was also an element of functional neurosis. It is known that encephalitis often produces "hysteriform" symptoms, the origin of which, according to Haškovec, is a subcortical morbid process.

A vestibular examination in the second case showed that cold irrigation of the right ear caused a rapid, mixed, vertical, upward and rotatory nystagmus to the right in the right eye, and a slow horizontal nystagmus to the left in the left eye; irrigation of the left ear caused a trembling nystagmus in both eyes to the left, followed by a divergent nystagmus. Turning to the right did not cause nystagmus but a falling to the left; turning to the left provoked nystagmus to the right in the right eye for thirty seconds and to the left in the left eye for twenty-five seconds.

"The deviation of the eyes to the side and upward, the spontaneous nystagmus, the sympathetic ptosis on the side to which the eyes were turned and the contralateral muscular hypotonia, with areflexia of posture, form a peculiar syndrome which, up to the present, we have encountered only in these two cases of atypical epidemic encephalitis. This is the syndrome to which we wish to call attention."

DENNIS, Colorado Springs, Colo.

ASTROBLASTOMAS OF THE BRAIN. PERCIVAL BAILEY and PAUL C. BUCY, Acta psychiat. et neurol. **5**:439, 1930.

This article reports the study of twenty-five cases of astroblastoma of the brain—one of the few groups of Bailey's classification of gliomas that has not been generally accepted. This type of tumor occurs predominantly in one of the cerebral hemispheres in adults, the average age of the patients on admission to the hospital being 38 years. The average duration of symptoms before operation was twenty months, and the average duration of life after operation seventeen months; i. e., the average total length of life from the onset of symptoms was thirty-seven months, which is in marked contrast to the expectancy of life for patients with astrocytomas (seventy-five months) and with glioblastoma multiforme (twelve months). Grossly, the tumors are diffuse, only rarely giving the impression of encapsulation. The fresh surface is pinkish gray with yellowish patches. Cystic areas are frequent, but hemorrhagic areas are not as common as in glioblastoma multiforme.

For microscopic study, metallic impregnation methods were not of much use. The best results were obtained by using Davidoff's modification of Mallory's

phosphotungstic acid-hematoxylin. Microscopically, the tumor shows an abundance of blood vessels, which are isolated from each other and have hypertrophied walls. In the walls of the vessels, the muscular cells can rarely be identified; the intima is thickened only occasionally, the hypertrophy being due to an increase in the number of pia-arachnoidal cells and infiltration by inflammatory cells (lymphocytes and plasma cells) of vascular origin. The perivascular neoplastic cells are clearly astroblasts.

Degeneration is common and begins between the vessels. Large amounts of fat are formed in these degenerative areas. The tumor must be differentiated from ependymoblastoma (to which it bears a close resemblance), glioblastoma multiforme and astrocytoma.

PEARSON, Philadelphia.

PSYCHOANALYTIC CONCEPTION OF DEPERSONALIZATION. SOPHIE MORGANSTERN. J. Nerv. & Ment. Dis. **73**:164 (Feb.) 1931.

Morganstern presents the case records of three patients suffering from depersonalization. In the first case the patient was only partially analyzed. He was a man, aged 48, who had had several attacks of depersonalization, all of which started with physiologic changes in his sexual life. This patient is said to have suffered from Oedipus and castration complexes which he could not resolve. The fear of castration was regarded as punishment for his onanism during childhood. In his second attack he gave up an enjoyable sexual attachment for a conventional loveless marriage. He showed proper insight into the abnormality of his feelings of unreality.

In case 2, a girl, aged 19, had suffered from the symptom-complex since the age of 11 years when she gave up masturbation because of the dreaded consequences. In this case the castration complex, the Oedipus complex, the question of sexual difference and the preoccupations of puberty were all offered as explanations of the depersonalization. This patient obtained complete relief from the analysis.

Case 3 was that of a girl, aged 17, in whom the sense of depersonalization was expressed in the repeated question "Who am I?" and who experienced sensations as if her soul and body had been separated. Masturbation with an adult man at the age of 7 years was an important sexual trauma in this case, which had homosexual features. In all three cases feelings of unreality occurred at crises in the sexual life. The author concludes the article with quotations from Janet, Ribot, Dugas, Moutier, Griesinger, Hesnard, Numberg and Federn with a variety of psychologic and psychoanalytic formulations. He considers that the distinction between reality and morbid sensation is the touchstone of the diagnosis of depersonalization. When this control is lost, schizophrenia is suspected.

HART, Greenwich, Conn.

THE OLFACTORY SENSE OF THE ADULT MEAL-WORM BEETLE, TENEBRIO MOLITOR (LINN.). J. MANSON VALENTINE, J. Exper. Zool. **58**:165 (Jan. 5) 1931.

The sexual response of the male meal-worm beetle, *Tenebrio molitor,* is definite, and the sensory equipment that is the principal means of guidance to the female is olfactory. Neither the optic nor the auditory organs are necessary for the successful discovery of the female. The male sexual odor is distinct from that of the female. The musky odor that is present when either sex is handled is probably merely an offensive, protective emanation. The organs of sense that guide the male *Tenebrio* to the female are located chiefly on the antennae. The antenna-less male is incapable of discovering a normal female. The male with normal antennae, but with the organs of McIndoo on the legs rendered ineffectual and those on the wings and elytra bases removed, reacts normally to the odor of the female. Circus movement in the reaction of the male to the odor of the

female is observed when one antenna is removed. A male with only the maxillary palpi amputated reacts normally to the odor of the female. The olfactory organs are located chiefly in the terminal four segments of the antennae. The sense organs through which *Tenebrio* receives the odor of food are identical with those operative in the perception of the odor of other individuals. Essential oils have an irritating effect, not only on the true olfactory organs, but likewise on organs normally receptive of other types of stimuli, and perhaps also on the respiratory tract. It is reasonable to suppose that the peg organs found on the four terminal segments of the antennae are operative in the response of the male to the odor of the female, and of both sexes to the odor of food. The slight superiority of the peg organ equipment of the male over that of the female may be correlated with the more aggressive mating behavior of the former. Experiments on beetles with ectomized palpi make it probable that the peg organ-like sensilla at their distal ends have a gustatory rather than an olfactory function.

WYMAN, Boston.

THE EFFECT OF SOME EXOGENIC FACTORS ON THE BLOOD-CEREBROSPINAL FLUID BARRIER. A. E. KULKOV, D. A. SHAMBUROFF and N. L. GARKAWI, Arch. f. Psychiat. **91**:658, 1930.

The results of experimental investigations of the effects that certain exogenic factors may have on the regulation of the passage of substances from the blood into the cerebrospinal fluid and the cerebral tissue are reported as follows: Rabbits were subjected to hyperthermia of from 41 to 43 C., following which the resistance of the barrier to trypan blue and to some iron salts and agglutinins, ordinarily not permeable, was studied. The first could not be demonstrated in the cerebral tissue at all. The second and third were found in the cerebrospinal fluid and also in the cells of the plexus and the leptomeninges. Introduction of neoarsphenamine and vaccines into the spinal canal brought about the passage of trypan blue into the cerebrospinal fluid. When large quantities of arsphenamine were used, some of the stain gained entrance into the tissue of the brain itself. The introduction of hexamethylenamin into the spinal canal facilitated the entrance of the stains into the cerebrospinal fluid. Furthermore, when there was an accompanying meningitis there was also a passage of these substances into the tissues of the brain.

The authors conclude that in studies of the passage of substances from the blood into the central nervous system two barriers should be considered: (1) the one between the blood and the cerebrospinal fluid, made up by the blood vessels and the plexus; (2) the one between the spinal fluid and the cerebral tissues proper, formed by the pia. Certain substances can influence the one without affecting the other.

MALAMUD, Iowa City.

POSTDIPHTHERIC LEVOGYRIC PARALYSIS OF THE EYES. JEAN SÉDAN, Rev. d'oto-neuro-opht. **8**:728 (Nov.) 1930.

This case report is one of a group of sixteen, published previously. It concerns a girl, aged 14, with obstinate postdiphtheric paralysis of lateral vision. She was unable to turn the eyes to the left, but convergence was not affected. There was slight deviation to the right, equal in the two eyes; diplopia was not present. General physical and neurologic examinations gave negative results. The Bordet-Wassermann reaction was negative. The patient's mother had had five other children and no abortions. Questioning elicited the history of a previous slight case of angina, and a culture from the throat showed the presence of Löffler's bacillus.

Energetic serum therapy was instituted without other effect than the production of rheumatic pains and cutaneous reactions. Improvement began after the use of 390 cc. of serum. The injections were stopped for twelve days, and

the improvement ceased. The serum was again given in doses of 20 cc., three times a week, until the patient had received a total of 620 cc., when ocular movements were restored to normal.

If the Wassermann reaction had been positive in this patient, and persistent search had not been made for the causal factor, antisyphilitic treatment might have been given, which would have resulted in the loss of opportunity to make the true diagnosis, since the throat meanwhile would probably have become free from the diphtheria organism.

DENNIS, Colorado Springs, Colo.

THE EDEMA AND NEUTRALIZING ACTION OF THE BRAIN IN VARIOUS SOLUTIONS. E. A. GOLDENBERG, Collected Papers, The Brain Institute, Leningrad, Bechterew Memorial Volume **3**:112, 1929.

The swelling of the isolated brain of a frog was studied in 0.7 per cent solutions of sodium chloride with the p_H varying between 1.27 and 12.80. In very acid solutions, in which the p_H is less than 3, the curve of swelling rises rapidly at first; then it slows down, the increase in weight in twenty-four hours reaching 40 per cent of the original weight of the brain. In very alkaline solutions, in which the p_H is 12 or less, the swelling of the brain proceeds rapidly, reaching 200 per cent of the original weight of the brain in six hours. In moderately alkaline and acid solutions the curve of fluid absorption rises very slowly during the first few hours, but increases after six hours, reaching an increase of from 45 to 75 per cent at the end of twenty-four hours.

The neutralizing action of the brain consists in the following: The p_H of the acid solutions in which the brain is placed changes toward alkalinity, while the p_H of the alkaline solutions becomes more acid, both converging toward a neutral zone. The most marked changes are noticed in moderately acid or alkaline solutions. When the hydrogen ion concentration is less than 3 or more than 11, the changes in the solution are very slight and in very acid or alkaline solutions they approximate zero. The neutralizing faculty of the brain is a confirmation of the universal property of tissue in regulating the hydrogen ion concentration of its biologic environment.

KASANIN, Boston.

INVESTIGATION OF THE CENTRAL NERVOUS SYSTEM BY POLARIZED LIGHT. M. MANDELSTAMM, Arch. f. Psychiat. **90**:805, 1930.

The value of the use of polarized light in histologic examinations of tissues of the central nervous system is discussed, and the results of such examinations are reported. The chief advantages in the method are the ease with which it can be carried out, the fact that it can be carried out soon after autopsy and the fact that it shows positive results in early degenerations when other methods fail. The method consists in examining under polarized light frozen sections, from 30 to 40 microns thick, that have been fixed in a diluted solution of formaldehyde, U. S. P. (1:10) for twenty-four hours. An arrangement for this examination can be easily attached to an ordinary microscope. Three types of constituents of nerve tissue can be thus studied: (1) myelin sheaths, (2) certain lipoid substances and (3) some substances lying freely in the tissues.

Mandelstamm reports the results of examinations of degenerations of myelin sheaths. The success of the method really depends on the same principle as in the case of the usual staining methods, that is, on the presence of certain lipoid substances in the myelin sheaths. Whereas definite loss or severe disturbance of these substances is essential before the lesion can be demonstrated by staining, the polarized light will demonstrate early changes in the physicochemical properties of the lipoid substances. Actually, in the cases quoted, sections that did not show any pathologic condition with the Marchi or Spielmeyer stains were found to be pathologic under the polarized light.

MALAMUD, Iowa City.

A Comparative Study of the Action of Bulbocapnine and Some Other Drugs in Producing Catatonic States. George W. Henry and Herman de Jong, Acta psychiat. et neurol. **5**:463, 1930.

This study is part of a series of investigations on the catatonia-producing properties of bulbocapnine. It is an attempt to determine the specificity of bulbocapnine in producing catatonia by comparing its action with that of other drugs, because if bulbocapnine has a specific action of this kind another step may have been taken toward the discovery of a metabolic toxic agent responsible for the catatonia of human beings. The effects of bulbocapnine were compared with the effects of cannabis indica, harmin hydrochloride, phenobarbital, sulphonal and somnifen. The effects of cannabis indica approach those of bulbocapnine more nearly than do those of the other drugs. The animal becomes drowsy, less active and indifferent to natural enemies, and loses desire for food and drink. Occasionally there is drooling, a tendency to maintain awkward postures and, with large doses, disturbances of equilibrium, swaying movements, ataxia and action tremors of the extremities. Negativism and complicated hyperkinetic symptoms did not appear.

When given in small doses, harmin caused drowsiness and a reduction of general activity. When given in large doses, it caused generalized tremors, hyperexcitability, hypertonicity, spasms and tonic clonic convulsions. Occasionally the animal tended to withdraw to a dark corner of the room. Small doses of the ordinary sedative drugs produce drowsiness and sleep; larger doses, disturbances of equilibrium, ataxia and hyperkinetic phenomena.

Pearson, Philadelphia.

Little Aids in Neurologic Diagnosis. R. Wartenberg, Nervenarzt **3**:594 (Oct. 15) 1930.

The author finds the following tests of aid in neurologic diagnosis: (1) Investigation of the skin sensibility by means of a pinwheel which is rolled with light pressure over the skin. It has an advantage over the use of a pin or a needle. (2) The Lasègue test by internal rotation of the leg. If this is done, Lasègue's phenomenon will become more definite. (3) Nerve stretching. In all cases of neuritis this produces pain. In this way Lasègue's phenomenon holds good for all nerves. (4) Testing of the skin reflexes, particularly the abdominal reflex, with the pinwheel. (5) Testing for the Babinski response after warming the foot. An indefinite Babinski sign becomes more definite after a warm foot bath, the action being similar to faradic stimulation of the other foot. (6) The glabellar reflex. A blow over the frontal or the nasal region produces bilateral contraction of the orbicularis oculi. This reflex is most definite after striking the region of the glabella. (7) Weakening of vibration of the upper eyelids as a sign of facial paralysis. (8) A position of abduction of the little finger in ulnar paralysis, especially in early and late symptoms of injury to the ulnar nerve. (9) Weakening of the pendulum swing of the arm while walking, as a symptom of a homolateral cerebellar lesion. (10) Unilateral lagging of the tongue in cortical lesions in the region of the face and arm centers and in lesions of the sensory fifth nucleus. (11) Weakness of the pulse in the foot in sciatic neuritis.

Hart, Greenwich, Conn.

Functional Reactions in the Embryonic Heart Accompanying the Ingrowth and Development of the Vagus Innervation. Philip B. Armstrong, J. Exper. Zool. **58**:43 (Jan. 5) 1931.

In embryos of *Fundulus heteroclitus* and *Fundulus majalis*, the embryonic heart beats for several days before there is physiologic indication that the vagus nerves have reached the heart. During this aneural stage the heart differentiates from a straight tube to an essentially adult condition. Vagus effects on the heart may

be obtained only after this differentiation is finished. The vagus effects are all negative, are abolished by atropine, and may be elicited reflexly by pressure on the head or in the region of the gills or on the tail of the embryo. The vagus response only gradually involves the various parts of the heart, so that five physiologic stages in the developing heart can be recognized. During the development of the physiologic effects of the nerves on the heart, there is no indication of a transposition of automaticity and conductivity from the cardiac muscle to the cardiac nervous system. Throughout all stages, the initiation of the beats remains myogenic and conduction of the beats, myodromic. The development of nervous responses in the heart of *Fundulus* is comparable to the development of swimming reactions in *Amblystoma* as described by Coghill. There is first a summation of responses to give the total response; this is followed by individuation, which in the heart of *Fundulus* is a more limited process than in the swimming reactions in *Amblystoma*.

WYMAN, Boston.

CONTRIBUTIONS TO THE PROBLEM OF CONGENITAL FEEBLEMINDEDNESS. M. HIELSCHER, Arch. f. Psychiat. **90**:731, 1930.

Following the studies of a number of investigators on anthropometric measurements and determinations of weight in normal children and the estimation of their progress over a period of years, the author has conducted similar studies in 400 cases of psychopathic and feebleminded children.

There were 269 feebleminded children (155 boys and 114 girls) and 131 psychopathic children (88 boys and 43 girls). The ages ranged from 6½ to 18 years. He found that the feebleminded children, both boys and girls, were shorter in stature than the psychopathic children of the same age. There was no appreciable difference in weight between these two groups. A comparison of stature and weight showed that in both groups there was none of the usual fluctuation in the ratio between growth and increase of weight that one finds in normal children, but an almost identical curve for both was obtained. This, however, should be checked further in a larger group of children.

It was found that the width of the body is greater in feebleminded than in psychopathic children. The index of length over width of the head is generally higher in the feebleminded children. These were mostly brachycephalic, hyperbrachycephalic or isocephalic, whereas the psychopathic subjects were mostly dolichocephalic, mesocephalic or brachycephalic.

MALAMUD, Iowa City.

SOME FACTORS INVOLVED IN THE REACTION OF INSECTS TO CHANGES IN LUMINOUS INTENSITY. SHOCK REACTIONS IN DINEUTES ASSIMILIS. LEONARD B. CLARK, J. Exper. Zool. **58**:31 (Jan. 5) 1931.

Dineutes assimilis, a gyrinid beetle, orients well in a beam of light. Each compound eye is divided into two parts, so each animal has apparently two pairs of eyes, a dorsal and a ventral pair. In all experiments the two parts of the left side were covered with a mixture of black Duco paint and asphaltum varnish. Specimens with one eye so covered, when placed in a beam of light on a suitable background, perform circus movements for a time, then go in a straight path diagonally across the beam. If, after the insects are oriented, the light is rapidly increased, they turn sharply from the source; if it is suddenly decreased, they turn sharply toward the source. These responses depend on light reflected from the background.

The elimination of circus movements is probably due to the adaptation to light progressing to such an extent that some of the ommatidia of the posterior portion of the eye are no longer stimulated after orientation. Rapid increase in intensity probably stimulates some or all of these ommatidia and the insect turns from the source of light. Rapid decrease in intensity probably causes cessation of stimulation in more of the ommatidia and the insect turns in the opposite direction.

WYMAN, Boston.

SURGICAL INTERVENTION IN MYELITIS COMPRESSION CAUSED BY ARACHNOIDITIS OSSIFICANS. L. PUUSEPP, J. Nerv. & Ment. Dis. **73**:1 (Jan.) 1931.

Osseous deposits in the arachnoid are of frequent occurrence. Virchow, however, showed that the number of modifications in the nervous system caused by osseous deposits in the arachnoid is so small as to be negligible. Leopold saw the principal cause in a disturbance of the circulation in the membranes of the cord, with a resultant irritative growth of connective tissue which begins in the vessels of the dura mater and penetrates the arachnoid, leading to solidification of the cartilage and later to ossification which is perivascular in distribution. Microscopic examination of the plates shows an ossiform center with an outer layer of connective tissue.

Puusepp presents four cases in which symptoms of compression of the spinal cord were noticeable and for which an operation was performed. Iodized poppy seed oil 40 per cent was found helpful when used in both ascending and descending directions. In all cases there was an absence of root pains, regarded by some authors as pathognomonic of tumors of the spinal cord. Compression of the spinal cord caused such symptoms as weakness of the lower extremities, paresthesias, increased frequency of micturition, diminution in sensibility, etc. Definite blocking of the flow of cerebrospinal fluid was demonstrated.

HART, Greenwich, Conn.

PERIPHERAL NEURITIS COMPLICATED BY MASSIVE COLLAPSE OF THE LUNG FOLLOWING TONSILLECTOMY. HAROLD V. DWYER, Arch. Int. Med. **46**:833 (Nov.) 1930.

An interesting question of etiology is presented by the case of an ironworker, who, a few weeks after recovery from lobar pneumonia, was admitted to the hospital with weakness and paresthesia in the arms and legs. He had a flaccid palsy of the lower extremities, and a diagnosis of peripheral neuritis was made. The patient had several sources of infection, including a septic mouth and diseased tonsils, which were treated by tonsillectomy and dental surgery. On the day after the former operation, atelectasis developed, and he died within a week. During the acute illness the weakness and paralysis cleared up. Dwyer raises the question of etiology: In spite of the patient's contact with lead, the author rejects this as a cause of the neuritis, because there was no laboratory evidence of absorption of lead. In spite of the fact that the neuritis improved after the infected teeth and tonsils were removed, he does not think that these foci of infection were the cause of disease. He suggests that the pneumonia preceding the onset of the neuritis was the responsible factor.

DAVIDSON, Newark, N. J.

GANGLIONEUROMA OF RETROPERITONEAL ORIGIN. REPORT OF A CASE, WITH BIBLIOGRAPHIC REFERENCES TO NINETY-THREE SIMILAR TUMORS. JOSEPH MCFARLAND, Arch. Path. **11**:118 (Jan.) 1931.

A white girl, aged 12, was observed to have a gradually enlarging abdomen. When asked about it, she complained of no pain or other disturbance nor could she in any way account for it. As the enlargement continued, the protuberant abdomen attracted attention more and more, and she was charged with being pregnant. A physician made a careful physical examination which resulted in the discovery of an abdominal tumor not connected with the uterus, which was normal and infantile. As the tumor had apparently grown as rapidly as the fetus for which it was mistaken would have done, malignancy was feared and an operation was recommended. This was performed in September, 1915, and a large nodular retroperitoneal mass was removed that weighed about 4½ pounds (2 Kg.) and was thought to be a sarcoma.

Since 1915, there have been about twenty-eight publications dealing with ganglioneuromas and reporting new cases, so that the tumor is much better known than when the case under consideration was observed.

WINKELMAN, Philadelphia.

GENERAL OBSERVATIONS ON SEA-SICKNESS AND THE LABYRINTHINE THEORY. T. GWYNNE MAITLAND, Brit. M. J. **1**:171 (Jan. 31) 1931.

Various possible causes for sea-sickness are analyzed, and new data are presented to support the author's conclusion that the major cause of this disturbance is dependent on stimulation of the vertical canals. If the head is placed in such a position that the vertical canals are stimulated, symptoms can be produced experimentally by rotation. This corresponds to the movement of the ship, namely, the pitch, which is considered the important movement in the production of this phenomenon. However, three rotations are necessary. These are not sufficient to produce nystagmus, and therefore explain why nystagmus is not present in sea-sickness. Eight rotations, however, do produce a period of rotatory nystagmus of six seconds, or vertical nystagmus of eight seconds, according to the position of the head, against fifteen seconds of horizontal nystagmus.

In regard to treatment, the author cautions against placing too much emphasis on any one form of therapeusis. The impression is gained that treatment should be considered as symptomatic and that there is no specific remedy.

FERGUSON, Niagara Falls, N. Y.

UNCONDITIONED AND CONDITIONED REACTIONS TO PAIN IN SCHIZOPHRENIA. LAURETTA BENDER and PAUL SCHILDER, Am. J. Psychiat. **10**:365 (Nov.) 1930.

In order to study the reactions to pain of patients with catatonic dementia praecox, Bender and Schilder used an induction coil provided with electrodes which were placed on the patient's hand. In this way it was possible to stimulate the skin with an electric shock; the apparatus also carried an electric light bulb which was used to establish a substitute stimulus to make possible the study of conditioning. They examined sixteen cases and noted a variety of responses: many of the patients squirmed and some showed stiffening of the body; in many cases there was lacrimation. However, in no case did the patient actively attempt to escape or to defend himself. Similar reactions could be obtained merely by flashing the light in the bulb, provided that this reflex had been conditioned by accompanying the first shock with lighting the bulb. Bender and Schilder believe that this experiment indicates that the individual's defenses are no longer integrated; that pain is appreciated only as a local phenomenon, and that the reaction is purely local. This disintegration of the response to pain is part of the patient's psychic disintegration generally.

DAVIDSON, Newark, N. J.

FURTHER OBSERVATIONS ON THE INFLUENCE OF TRANSPLANTED EYES ON THE DEVELOPMENT OF SPINAL NERVES IN AMBLYSTOMA PUNCTATUM. T. C. NUSSMANN, J. Exper. Zool. **58**:21 (Jan. 5) 1931.

When an optic vesicle of *Amblystoma punctatum* was transplanted into the right limb site of another embryo whose right limb rudiment had previously been removed, the brachial nerves approached and passed beside the transplanted eyes, finally ending in muscle or in other peripheral structures. When a limb regenerated, the brachial nerves innervated it normally. Spinal nerves did not innervate the transplanted eyes. When an eye only was found in the limb site, the spinal ganglia showed a hypoplasia similar to that which occurs when a limb is removed and no other transplant placed in the limb site. When a limb regenerated, the

ganglia showed normal development. The transplanted eye had no effect on the development of spinal ganglia. This is further proof that the development of spinal ganglia is in large measure dependent on the connections of their nerves with end-organs. The fact that spinal nerves made no connections with the transplanted eyes suggests that there is a capacity for selectivity or specificity that limits the potentialities which nerves have for making connections with end-organs. This specificity may be better explained as galvanotropic than chemotropic.

WYMAN, Boston.

THE KNEE JERK AND ITS VARIATION—AN INDICATION FOR SYMPATHETIC RAMISECTION OR TRUNK SECTION. N. D. ROYLE, Brit. M. J. **1**:11 (Jan. 3) 1931.

A description of the knee jerk is given, with directions for the proper method of its elicitation. In this connection the author emphasizes the importance of having the patient in a reclining position, with the neck and shoulders supported and the limb to be tested hanging over the end or side of the couch or table. The nature of the response, the rate of return to the normal position and the number of oscillations give one an approximation of the tonus present.

In Raynaud's disease, Hirschsprung's disease and Buerger's disease and in certain other vasospastic conditions, the knee jerk shows an increased excitability and a prolonged relaxation time, with few or no oscillations when coming to rest. Thus, the nature of the knee jerk in these conditions may be used as one index for operation. The knee jerk in fifty medical students was examined as a control. It was normal in all save three. Two of these were long-distance runners and had hypertonia, and the other had had poliomyelitis.

FERGUSON, Niagara Falls, N. Y.

THYROTOXICOSIS FOLLOWING SUBTOTAL THYROIDECTOMY FOR EXOPHTHALMIC GOITER. W. O. THOMPSON, A. E. MORRIS and P. K. THOMPSON, Arch. Int. Med. **46**:946 (Dec.) 1930.

After studying the postoperative history of almost 200 patients who had had a subtotal thyroidectomy, Thompson and Morris found a persistence of thyrotoxicosis in 20 per cent; only 1 per cent showed a recurrence of hyperthyroid manifestations. The authors believe that the persistence of the toxic condition is due to the removal of too little tissue at the time of operation. The preoperative use of iodine is not a factor, and they could not find before operation any way of predicting which patients would not get well. They suggest iodine and roentgen therapy in cases of persistent thyrotoxicosis and report that this treatment will succeed in bringing about clinical improvement in most cases; patients who are not improved by these measures should undergo reoperation, and more tissue of the thyroid gland should be removed.

DAVIDSON, Newark, N. J.

SYMPATHETIC RAMISECTION IN SPASTIC PARALYSIS: END-RESULTS OF SIXTY CASES. W. B. CARRELL, J. A. M. A. **96**:849 (March 14) 1931.

Sixty cases of lumbar ramisection and twenty-three cases of cervical ramisection were studied in an effort to estimate the effect of the operation. An increase in postoperative temperature was recorded in 40 per cent of the cervical and 100 per cent of the lumbar cases. No changes in the character of the reflexes or in contractions were observed, but in cases presenting the most improvement, a relative increase in joint range was noted. An improvement in coordination and equilibrium is recorded. In twenty-three cases other operations, in addition to ramisection, were performed; the results of the latter procedure are evaluated separately. The severity of the lesion and the mental age of the patient were important factors in the improvement. The author, with Dr. Fred T. Rogers,

removed the left sympathetic trunk in eight goats, and followed this with decerebration after from 60 to 120 days. The previous sympathectomy did not influence the rigidity of the decerebrated specimens. The work of other experimenters in the field is reviewed.

JENKINS, Indianapolis.

WHO ARE THE CRIMINAL INSANE? RUTH F. YEOMANS, Ment. Hyg. **14**:672 (July) 1930.

After an analysis of the case records of fifty persons with mental disease who had committed criminal acts, Miss Yeomans observed that the civil and economic distribution of this class was wide, and that the problem of the mentally diseased offender was the problem not of any group, but of society as a whole. She suggested that early mental testing be applied generally to recognize the defective child and to promote his readjustment to society as early as possible. A psychiatric study of the group of criminals with mental diseases disclosed a high incidence of psychoses related to alcoholism, more than one fourth of all the cases in this study being diagnosed as some form of alcoholic psychosis. This complicates the problem of handling criminals with mental disease, for the mental hospital cannot retain the alcoholic patient after his restoration to sanity, and his release will again precipitate him into the inevitable cycle of drinking, offending and being arrested. The author suggests the erection of institutions for chronic inebriates. Of the patients included in this study, one-third made a more or less satisfactory adjustment in the community after discharge from the hospital.

DAVIDSON, Newark, N. J.

DISTURBANCES OF DEGLUTITION IN NEUROPATHOLOGY. LEDOUX, Rev. d'oto-neuro-opht. **8**:677 (Nov.) 1930.

This paper consists of introductory remarks that were made while showing a moving picture of the act of swallowing. Of the three phases of deglutition, the pharyngeal is the most important from the point of view of neuropathology. The most important element in the pharyngeal phase is elevation of the larynx and opening of the mouth of the esophagus. The only means of observing this elevation is by an analysis of the movements of Adam's apple. This is done by having the patient drink liquids in order that there may be no participation of the superior constrictor muscle and to fatigue the elevator muscles. Claudication in the elevation of Adam's apple is an early sign of difficulty in swallowing and occurs long before actual obstruction is present. Ledoux believes that it is possible to dissociate early the involvement of the various muscles concerned in deglutition and to determine which nerves are injured, either in the neuraxis or in the periphery.

DENNIS, Colorado Springs, Colo.

THE EFFECT OF EXPERIMENTAL EXCISION OF ONE EYE ON THE DEVELOPMENT OF THE OPTIC LOBE AND OPTICUS LAYER IN LARVAE OF THE TREE-FROG (HYLA REGILLA). II. THE EFFECT ON CELL SIZE AND DIFFERENTIATION OF CELL PROCESSES. O. LARSELL, J. Exper. Zool. **58**:1 (Jan. 5) 1931.

Excision of one eye in the tree-frog, *Hyla regilla*, after connections with the optic lobe had been established and after optic function had been demonstrated, resulted in failure of the affected optic lobe to develop to normal size or to proliferate the normal number of cells. The neuroblasts of the eighth and ninth layers were reduced in number. They also failed to grow to normal size or to show the normal development of dendritic processes. These results appear to be caused by the absence of the normal functional stimuli that reach the optic lobes in the normally developing frog or by the absence of factors directly attributable to these stimuli.

WYMAN, Boston.

THROMBOPHLEBITIS OF THE CAVERNOUS AND INTERCAVERNOUS SINUSES: BILATERAL ORBITAL THROMBOPHLEBITIS CONSECUTIVE TO PERITONSILLAR ABSCESS. EEMAN, Rev. d'oto-neuro-opht. **8**:744 (Nov.) 1930.

This case report emphasizes the fact that peritonsillar abscess is a dangerous condition. The patient showed a typical picture of cavernous sinus thrombosis and died four days after admission. The venous connections are described as follows: "The orbital veins communicate in front with the facial veins, in back with the cavernous sinus. The cerebral veins communicate below with the veins of the pterygoid plexus." Infections of the face, mouth or pharynx, especially of the nose and eyelids, are the common cause of thrombosis of the cavernous sinus. The prognosis, which is grave, and the treatment, usually ineffective, are discussed.

DENNIS, Colorado Springs, Colo.

CEREBROSPINAL FLUID OF PREMATURE INFANTS. JEROME GLASER, Am. J. Dis. Child. **40**:741 (Oct.) 1930.

Using a small hypodermic needle for the lumbar puncture, Glaser studied the physical and chemical properties of the spinal fluids of 170 premature infants. He demonstrated that xanthochromia is normal in these cases, and, after investigation of the van den Bergh reactions, he concluded that this physiologic xanthochromia of the premature infant represents an immaturity of the blood-spinal fluid barrier. In many cases a hazy fluid was found which, Glaser believes, was due to the presence of a few erythrocytes and indicated acute meningeal congestion.

SPINAL DRAINAGE IN ALCOHOLIC DELIRIA AND OTHER ACUTE ALCOHOLIC PSYCHOSES. HARRY GOLDSMITH, Am. J. Psychiat. **10**:255 (Sept.) 1930.

If a lumbar puncture is performed in patients with acute alcoholic delirium, with drainage of 40 or 50 cc. of spinal fluid, they will show rapid mental and physical improvement. This has been the observation of Goldsmith, who adds that this treatment is most effective when instituted within twenty-four hours after admission to a hospital. Harmful sequelae do not occur and the patient's stay in the hospital is reduced. In almost 80 per cent of the patients in whom drainage was done, recession of acute mental symptoms occurred within a week.

BEHAVIOR PROBLEMS IN CHILDREN WITH SPECIAL REFERENCE TO DELINQUENCY. IRA S. WILE, Am. J. Dis. Child. **40**:1076 (Nov.) 1930.

Deploring the tendency to attribute juvenile delinquency to some simple, single agent, such as mental defect, endocrinopathy or parental incompetence, Wile emphasizes the complexity of factors that must be understood before one can gain insight into the problem of misbehavior. Feeblemindedness has, he believes, been overemphasized; he quotes Healy and Bronner who have reported that only one juvenile delinquent in ten may be considered mentally deficient or psychopathic. It is therefore necessary to manage behavior problems specifically, studying each case as an independent complex and avoiding generic diagnosis and treatment.

PSYCHOSES DUE TO DRUG ADDICTION. ALPHONSE RODIET, Arch. internat. de neurol. **3**:373 (Nov.) 1930.

After studying many cases of mental disease due to drug addiction, Rodiet concludes that the outlook is bad and the condition virtually incurable. Regulation of the sale of narcotics has failed as a prophylactic measure. The defects in character and the handicaps in environment that allowed the addiction to occur in the first place will probably always exist for the individual patient. Treatment by absolute and sudden withdrawal is barbarous and dangerous, while gradual reduction in dosage is slow, impracticable and ineffective.

DAVIDSON, Newark, N. J.

Society Transactions

THE NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY

Regular Meeting, Dec. 9, 1930

DR. MICHAEL OSNATO, *in the Chair*

A HEBEPHRENIC SYNDROME. DR. LELAND E. HINSIE.

History.—The syndrome in this patient appeared shortly after puberty and was characterized by the following phenomena: (1) steady, progressive withdrawal from reality, growing out of a shut-in childhood; (2) regression to early Oedipus manifestations (strong attachment to father; replacement of mother by self); (3) autoeroticism; self-interests exclusively; appearance of feelings of superiority; (4) later transitory states of complete depersonalization; identification with the impersonal universe; feelings of tremendous greatness in size; (5) complete subservience to fantasies, with periods of insight; loss of capacity to distinguish fantasies from realities; sometimes a distinct preference for fantasies; sometimes an aversion; (6) inappropriate emotional reactions: "spontaneous" laughter; anger evoked by pleasant thoughts (parathymia); joyous experiences accompanied by complaint of paraminia; objective apathy; subjective interestedness; (7) unstable, transitory attachment to girls; (8) disturbances of association; lack of logical sequence in thought, neologisms with intense affective investment; (9) fluctuation of the foregoing processes; lack of permanency or fixation; psychotic phenomena referable, in general, to the pregenital phase of development, with a richness of "archaic manifestations"; correlative to this, reversion of mentation to feeling, concreteness and perception (law of Domarus), after a short excursion into the realm of reasoning, differentiation and abstraction; (10) frequency of occurrence of insight, with attendant annoyance to the patient; no permanent loss of insight; (11) clarity of sensorium at all times; (12) lack of fundamental "projection" of thoughts or feelings; occasional, ill-defined ideas of reference; (13) absence of hallucinatory experiences, and (14) of known physical disturbances to be correlated with the aforementioned psychotic changes.

Course.—The patient was under a psychiatrist's care continuously for one and one-half years. More or less intensive and extensive investigation was made into all psychic recesses, the procedure having been largely "cathartic" in nature, with explanations ordinarily coming from the patient. From the point of view of the patient, the psychiatrist was exasperatingly passive with regard to interpretative technic.

Gradually the symptomatology disappeared and partial familial, social and industrial adjustments took place. The patient eventually recognized that she had only a few mild psychotic phenomena remaining and was disappointed because she had been "deprived of the best way of adapting myself." She attempted several forms of adaptation to reality (parties, piano lessons, dancing, together with a few incomplete heterosexual affairs) but they had not been fully satisfying. For the last six months she had been engaged in testing reality, though she was not certain that she preferred it. Her interests were distinctly more objectivated than they had been before the psychotic period, but there remained an uncertain, unstable adjustment.

For experimental purposes she was left completely to her own resources on the completion of treatment. Perhaps it would have been better to have had her followed steadily by some other agency, such as a social service worker.

Physical Status.—There had been an uneventful physical growth in early childhood. There was some pituitary hypofunction, manifested principally in obesity, but also in skeletal growth, beginning before puberty. Otherwise there was no outstanding physical disorder in the history. Secondary sexual growth had been uneventful.

Orientations of the Training and Practice in Neurology and Psychiatry. Dr. Adolf Meyer.

The problems of orientation and reorientation of the practice and teaching and research in psychiatry, neurology and mental hygiene can be brought home with the frequently recurring questions: To whom had I best refer a patient with personality difficulties or disorders? Where should I send a promising student for his training in psychiatry, neurology and mental hygiene? How should I advise a university to organize the pertinent departments?

Neurology and psychiatry in the eighties and nineties showed a remarkable simplicity. The neurologist was armed with his electrotherapeutic outfit and, perhaps, hypnosis and rest cure, and had as his intellectual diet the neuron theory and the beginnings of experimental neurology; the psychiatrist was equipped with his institutional and court practice, limited, so to speak, to the problem of "insanity." This simple, uncomplicated arrangement has given way to a very different alinement.

Neurology has been both enriched and depleted in many directions: syphilology, infectious diseases and immunity have gone their own way, and psychopathology has become affiliated with psychiatry, so that its most virile core is today formed by neurosurgery and a new type of neurophysiology and, in the main, a strictly organic reflexology.

Psychiatry has gone beyond the onset of disorders to their origins and it has drawn within its sphere the minor psychoses and psychopathology generally and, also, the infantile defects.

Mental hygiene has arisen, with perhaps excessive promise and propaganda, but with a healthy core in its approach from the angle of health and of prevention of all those conditions which, though not actual factors of the ill health, might work against the health, happiness and efficiency of the individual and the group.

In all of these fields and in the many new specialties, there is the need of a sound medical training, but there is also a reciprocal appeal for reasonable orientation in neurology, psychobiology and psychopathology as one important supplement of all medical practice, and for a recognition of the need of standardization of training and organization for special work in psychiatry, mental hygiene and neurology.

The most urgent need is a frank recognition of a real autonomous field for the (mentally integrated) personality functions and personality difficulties, and a frank recognition of the psychiatrist for his services in psychiatry and not merely for some incidental and possibly extraneous factor, such as his interest in infections of the brain or other parts, such as the endocrine glands. He must have these latter interests, but if he does not above all have a mastery of the personality functions and personality difficulties and of the psychobiologic processes, he is not a psychiatrically trained physician.

In mental hygiene one gets into a field in which one must guard against being perhaps too ardently guided by professionalism, but the only way to avoid a kind of "bootlegging" by uncontrolled nonprofessional "clinics" will be to establish standards of work making clear the contrast between a sphere of ordinary trained common sense and the sphere of assumption of responsibilities with the sick and those in need of competent medical examination and attention.

Neurology and psychiatry work under the handicap of dealing with a structure that is organized for a lifetime, a record and integrator of the life history and the life experiences of the personality. This makes psychiatry with its demand of personality work a definitely specific field. The practical results with patients in both neurology and psychiatry can be startling, but the prospects of a considerable contribution to mankind might be discouraging, if psychiatry were not cultural and academic in the best sense of these words, the most vital balance in a transformation of a view of man from too exclusive preoccupation with man in the light of eternity to one of due attention to the nature of a biologic organism. Thus, psychiatry has to work with integration of the lives of young, adult and older generations and with a real concern for the next generations, with a sound adjustment of concern for whole and part, a balance of individual aims and means, and of freedom and solidarity.

Psychiatry and neurology and mental hygiene had best form a special group, but one made up of reasonably independent departments for their intensive work, with liaison service in all the other departments of medicine. Arrangements for service, teaching and research, in the order of sequence, are urged as the sine qua non of a sound foundation and development as a university division.

HAVE PSYCHOLOGIC CONCEPTS A PRACTICAL VALUE IN NEUROPSYCHIATRY? DR. EDWARD A. STRECKER.

Psychologic concepts may be discussed from the standpoint of practical therapeutics in the problem of the neuroses. However eclectically minded the physician may intend to be, the human mind is so constituted that he will follow some particular theory in the treatment of the neuroses with more confidence than any other theory. When confronted by an actual case, the physician after his preliminary examination will finally face a situation in which he must select a definite method of therapeutic attack. If he follows the father of medicine, Hippocrates, to the extent that he believes that the brain is the organ of the mind in every sense of the phrase, then he will think of the patient in terms of ultramicroscopic pathology of the brain. If, on the other hand, he is a believer in the theory of focal infection, he will follow this theory to its logical therapeutic conclusion. If, however, he is convinced that the clue to the neurosis and its treatment lies somewhere in the hidden mental life of the patient, then he will employ some form of treatment based on psychopathology. I shall not discuss the various schools of psychopathology but shall lay down a minimum of psychopathologic belief that I regard as necessary to the treatment of the neuroses. Among these conceptions the following are emphasized: (1) The so-called "complex," by which is understood an imperative demand for expression in action of a group of ideas held together by a strong emotional bond, is an actuality. If the complex is at odds with the ego of the individual or the code of society it must inevitably produce the conflict, which may often result in the production of nervous phenomena. Other mechanisms that are often employed and that produce neurotic symptoms are: regression, rationalization, repression, dissociation, projection and identification. Furthermore, if the clinician wishes to gather the therapeutic fruits of psychopathology, he must be willing and able to believe that mental conflicts may be and frequently are converted into physical sensations and symptoms and that the way to remove such sensations and symptoms is to trace them back to their beginning. Again, it must be believed that a so-called sense of inferiority is an actuality and not merely an armchair philosophy, and that in a given instance it may result through poor compensation in the development of a neurosis. (2) Even though the maximum that may be hoped for from psychopathology is a frank realization by the patient of the nature of his conflicts, yet such realization in itself has great therapeutic value. In fact, it seems almost a dictum that if a person is clearly aware of the nature of his conflicts he can scarcely develop a neurosis.

DISCUSSION ON PAPERS BY DRS. HINSIE, MEYER AND STRECKER

DR. BERNARD SACHS: I take it that Dr. Meyer's object was to discuss the development of neurology, and its relation to general medicine, to psychiatry and to mental hygiene. This question of relation is one that is uppermost in the minds of neurologists the world over, as I had occasion to learn during the past year. There is hardly a country in Europe — Germany, Austria, Switzerland, Great Britain and, also, possibly, France — in which neurologists are satisfied that justice is being done them. They deplore the fact that a knowledge of neurology is not required for a medical degree in Germany. There are only two full professors of neurology in the whole of Germany. It is not required in Austria or in Switzerland, and in Great Britain the complaint is that neurology is not fully recognized in the universities or in the larger hospitals. The dissatisfaction is not a personal one, but it has a great deal to do with the general estimate formed of the relation of neurology to the practice of general medicine, and especially its relation to psychiatry.

It would be easy from personal experience to endorse some of the statements that Dr. Meyer has made. We have all passed through that era in which, first of all, we were called on to assist, or at least to participate, in the development of the neurologic science which, forty or fifty years ago, was a science concerned with the development of distinct clinical entities that could be ascribed to the central nervous system. It was the period during which, for instance, the diagnosis of myelitis and of cerebrospinal syphilis, and the discussion of localization of the spinal and cerebral functions were very much in the foreground. These questions were so satisfactorily settled that at the present time they need not be considered.

At present we are especially engaged with the cause, the general development and the cure of the so-called neuroses and psychoneuroses. In the search for the proper treatment of the neuroses, it was soon made evident that the neurologist could not lay claim exclusively to the neuroses, that they were disorders that spread into the psychiatric field. There are few pure neuroses; most of them are psychoneuroses. The study of the neuroses was naturally begun first by the neurologist; it was subsequently taken up by the psychiatrist.

No one is able to draw a hard and fast line between the neurologist and the psychiatrist. We are all neuropsychiatrists. At least, neurologists think and feel that they must know something of psychiatry, and I hope that the psychiatrists feel that they must know something of neurology. The two belong together and cannot well be separated.

In the development of the study of the neuroses and psychoneuroses, various trends have been established, various schools have sprung up, and one is often asked to become a follower of one or the other school. All that I ask, is that one thing to which Dr. Strecker referred be allowed, namely, that, regardless of the explanation sought for the neuroses or psychoneuroses, one should pay distinct attention to what Dr. Strecker calls "logical" thinking, and should avoid a good deal of the "emotional" thinking. One can afford to follow any school, provided one remembers that logical thinking is the only sort of thinking permissible in practice. Whether in one field or the other, one must regard the relation between cause and effect that is allowed in all sciences, consider the premises from which proper inferences can be drawn, and not take assertions on faith because one prophet or another has made them. I will make a confessio medici: I believe in the most careful and rigid mental analysis, but I am not willing to accept any doctrines that apply to mental disease or mental phenomena that do not fully answer every possible logical demand. The progress that has been made in neurology, and in psychiatry too, has been in the theories and in much more careful study of the behavior problems and personality problems. One must study the individual. There is a far better means of study than before in regard to social relations, family relations, and so on. There are many forms, "mechanisms" and "complexes" now that were not known before. Some of the complexes have become very boresome. There is one very popular one, the inferiority complex.

Any one even ordinarily modest is accused of having an inferiority complex. I find the superiority complex much more frequent and a much more distressing social and medical condition. If you ask what I mean by remaining logical, let me single out one subject. A great deal has been said about the subconscious. Any amount of interesting information has been furnished in regard to the subconscious, but the protest that I make is that because much information has been given regarding the subconscious one must be careful in the logical deductions one draws, and because certain facts have been demonstrated as occasionally occurring in the subconscious, this by no means justifies the generalization that all actions are controlled by the subconscious. Let us draw logical deductions from carefully established facts.

This leads me to the movement for mental hygiene mentioned by Dr. Meyer with which I am more or less directly connected. This movement is directed generally toward keeping normal minds normal, and preventing, as far as possible, the development of mental disease. I have always considered the term "mental hygiene" unfortunate. How simple "mental health" would be. The word "hygiene" in connection with the mind carries all sorts of ideas to some, and has perhaps been confusing. The object has been to secure these two ends, the preservation of the normal personality and the prevention of the diseased personality and diseased mind. I am anxious that we should not be led along false paths in the further development of this movement. It is necessary, particularly as the laity is so interested in the mental hygiene movement, that we do not give one-sided or imperfect information, particularly in connection with sex. The layman should not be taught that sex is the only thing that dominates this world and our lives. It is an important factor, but to most of us it is not the dominating factor. The mental hygiene movement has to be rescued from the idea that the future of mental hygiene lies largely in the direction of that special branch which, whatever its object may be, eventually leads to the discussion of some sex problem.

DR. SMITH ELY JELLIFFE: While I was listening to the two addresses, one of Kipling's ballads insisted on emerging from the preconscious. The first stanza runs as follows:

> "When 'Omer smote 'is bloomin' lyre
> He'd 'eard men sing by land and sea
> An' what he thought he might require
> 'E went an' took — the same as me."

And so, what shall I take from the richness of the feast just spread before us? Dr. Meyer has reminded us to use "common sense" in our thinking about the numerous problems involved. He has asked to whom he should send his hypothetical patient for advice and treatment. My common sense answer, with a Shavian tongue in my cheek, is, "Send him to me!" And if asked what I can do for the patient, my first answer would be to state what I would not do. In the first place, after looking over the human machine I would not knock out a spark plug; I would not tinker with the carburetor; I would not saw off the transmission, nor would I take off a wheel or two. I doubt very seriously if I would attempt to straighten up a septum deviating $\frac{1}{32}$ inch from that monstrous abstraction "the normal," nor clean out those so-called "sink holes" of iniquity, the tonsils. The teeth likewise might be left alone, and the cervix left unamputated and I would permit the patient to keep his entire intestine intact, Lane's kinks or other anomalies to the contrary, even if I looked on with irritated silence at the misuse of those persisting remnants of the practices of Sodom, the 'Cult of the Clyster' with its innumerable delusions of 'sewer' cleaning. If my common sense did nothing else for Dr. Meyer's patient, it at all events would not return him mutilated beyond any semblance of a human being.

Turning to Dr. Strecker's equally pertinent question regarding the minimum of agreement that I can find in all of the various aspects of therapy, I would return to my Homeric mood and ask that you come with me and join the Greeks on a medical Mount Olympus and listen in on a broadcasting program. Aesculapius

is there in the rear, Hippocrates, Democritus, Heraclitus and the rest. Plato is over there to the left with a dunce's cap on his head, being punished for his defection from the Homeric conception that the soul belongs to the entire body, the "organism as a whole" that we have heard so much about this evening. Even Aristotle, a typical Oedipus, is jeering at his father for his "disembodied soul" for, says Aristotle, the soul is to the body as "axness is to the axe," or as "vision is to the eye." And now one hears the announcer from the earth beneath: "You are now listening in on station W O O F, W O O F, Clarissimus Galen in Rome, 200 A. D., is lecturing on hysteria. W O O F, W O O F." "Gentlemen, this patient I am showing you has had pains here, and disturbances there, in the back, in the arms, in the kidneys, all over the body and now she has a lump in her throat. She cannot breathe; she is all undone. This gentlemen, is a classic example of globus hystericus." And may I remind you what old fogies those early Greeks were? Especially was this true of Hippocrates when he took up with the nonsensical theory of Democritus, who 600 or more years ago taught that this hysteria was due to the wandering uterus which, like a wolf seeking to be impregnated, wandered now here, now there, until finally it got up into the throat. Now, fellow practitioners, come with me into my laboratory and I will show you what foolish notions these ancient graybeards taught us. Look into this cadaver; notice these broad ligaments. How can the uterus move as Democritus taught? And so, anatomy and chemistry and physics all demonstrate the foolishness. . . .

At this one hears a Rip Van Winkle thunderbolt and the Olympian medicos are splitting with laughter, especially Democritus, the laughing philosopher. "The dolt, the static fool," one hears him shout. "He thinks I was talking about the actual uterus when I was referring to the psyche, the soul, which our friend here, Homer, insisted, even 600 years before my day, animated the entire body. It is not confined to the head, even though a brain is needed for logical thinking. I tried to show that the creative spirit in man might find an outlet through any or every organ of the body."

Here Olympus passes from our vision, and I return to say of the principle of a minimum requirement that I would do my utmost for the hypothetical patient of Dr. Meyer to release those forces within the body and arrange those forces without the body to permit the greatest freedom to the creative spirit, the psyche, as old Democritus taught 2,500 years ago.

Dr. J. Ramsay Hunt: To all who are interested in the future development of neurology and psychiatry, Dr. Meyer's paper will be of the greatest service. He has been an important leader in this field during many important changes which have taken place in the last quarter of a century and his neurologic background fits him in a peculiar way to present both the neurologic and psychiatric points of view. It must not be forgotten that in the development of psychiatry, what is termed neurology has played an important rôle. Neurology grew out of the study of internal medicine, and until recent times the neurologist was the psychiatrist of the general hospital and of the community, and the psychiatry of the present day owes much to this association. The psychiatrist, on the other hand, was confined to institutional work, and his training was almost exclusively in the major psychoses. It is only in the last decade or two that selected men from this group have engaged in teaching, research and practice on the same terms in the community as did the neurologist. This has taken place because of the recognition of the great importance of the specialized psychiatric point of view in medicine, law and social science.

There have been certain differences in the points of view of neurology and psychiatry, but the development of psychiatry, broadly speaking, has been in the hands of both groups. Indeed, the field of medicine has also participated in the development of psychiatry, and the first important studies in this country were made by Benjamin Rush, a physician of Philadelphia, who wrote the first book on disorders of the mind published on this continent.

While this subject is under discussion, it is of interest to recall some of the more important neurologic influences in psychiatry. Hughlings Jackson and his

views on the evolution and dissolution of the nervous system played an important rôle not only in neurology but also in the psychiatry of that period. At the same time, the psychogenic conception of hysteria emanated from the neurologic clinic of Charcot. Another most important influence from the neurologic side is the work of Sigmund Freud, who made studies on the clinical material as it came to the office of a neurologist. There he made those fundamental analyses that have had such an activating effect on the psychology of today. Indeed, a great deal of the ferment and movement in modern psychiatry are based on his studies. Also, our own Morton Prince was a pioneer in the field of abnormal psychology and the analysis of mental mechanisms. There have been many others, showing the great activity of men of neurologic training in this large and important field. Neurology stands on very firm ground in claiming recognition in the modern psychiatric movement.

I regret that Dr. Meyer has not made more of neurology and the neurologist in his orientation, but still keeps neurology somewhat separate in his own mind as a field that is concerned with the study of fragments and parts of the nervous system. The point that I wish to make is this, that the neurology and psychiatry of today are gradually merging, and that those who are leaders in these fields should, as far as possible, assist this process, which is not always an easy one.

In my judgment, the ideal psychiatrist of the future will be a neuropsychiatrist, that is, a man trained not only in neurology and internal medicine but also in psychiatric methods and experience. Within this broad field there will, no doubt, be numerous specialists devoting themselves intensively to a particular field, but they will be men of similar training and background. I believe that the psychiatrist should never get away from neuropathology and neuro-anatomy. Who knows what may happen in a few decades in the field of chemistry in relation to the cells of the brain, and what influence this will have in building up a new psychiatry?

Today psychiatry has invaded the jail, the school and the market place. We are passing through this period on the various slogans of mental hygiene, but we shall certainly swing back to consideration of the central nervous system as the positive source of psychiatric disorders, and the men who will be the foremost teachers and investigators in that field in the future will be neuropsychiatrists, that is, men who combine all that is best in the two disciplines.

Dr. Israel Strauss: I think that Dr. Hunt has not really understood Dr. Meyer's position, because Professor Meyer started out by alluding to the work of Pawlow and other physiologists. He also referred to the possibility of physics and chemistry affording enlightenment on the processes of the mind, although he thought that it would not be possible to connect the two. Dr. Meyer has always been talking, as he did tonight, of the unity of the organism as a whole, so he cannot logically — I am speaking of logical as opposed to emotional thinking — separate the organic from the functional. At the present time we may not be able to connect them as well as we should, but certainly the attitude of science, if it is going to advance, must be to connect the functional with the organic.

Dr. Meyer has also spoken of the teaching of psychiatry. Years ago, I advocated that and I still do, and I go further and say that the student in high school should be taught psychology. I mean the psychology that has to do with the personality. The difficulty has been, however, in finding the psychiatrists to act as teachers. I do not think that the problem is so much to get the schools and centers, but the instructors who can teach with logical and not emotional thinking. You have seen examples of emotional thinking this evening. You have various needs and clashing interests, and I think that Dr. Meyer will discover that his difficulty is to find what point of view he is going to use in instructing the students. One psychiatrist will want to teach from the standpoint of behaviorism, and another from the standpoint of psychoanalysis. In time there will unquestionably be a union on common ground, and in the meantime, the physician and student should be taught as much as possible.

Dr. Paul Schilder: In modern psychiatry, the point of view emphasized is that of personality. Personality for us is not abstract, but concrete. We observe

that all events in a person's life have some connection with each other. We see and we know, and this is one of our great advances in psychiatry, that early events never disappear completely. They retain their value and influence. Nothing is forgotten. Everything remains in the personality. Our conception of personality is therefore dynamic. We see that these early events shape what happens in a person's future, and that the manner in which he lives will be determined by everything that has happened in his life.

We also have a changed attitude concerning constitution. We believe in its existence, but we do not think that it is a rigid thing; its expression is considerably altered by the emotions of the individual, and the events in his life. Therefore our chief aim is to understand the individual, and to understand why the individual's constitution has taken on a particular form. If we understand this, we can help his constitution to alter its shape. This is the aim of psychotherapy. We cannot change his constitution, but we can give the individual the opportunity of using the constitution as it fits the personality and the social aims. Of course, what we can do is limited. We cannot go very deeply into the organic field. The organic field can be influenced by the factors that shape the constitution, these being psychogenic and organic at the same time. They come out in psychotherapy as libidinous factors and factors of psychic energy. These interefere with the constitutional and organic factors. In short, the whole life of the neurotic patient, and the psychotic as well, can be reestablished under the influence of psychotherapy, which deals with the problems of personality, and tries to give back to the individual the capacity to live out his constitution and his own personality. I think that this is the aim of what we may call modern psychiatry, to help the individual to develop into himself.

DR. ADOLF MEYER: I wish merely to express my thanks to the audience, and to assure you that the comments that were made, whether corroborative or otherwise, will become part of the flux of further development, which Dr. Jelliffe has voiced as an old concept and an old theory of the Greeks, and one that will last forever.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 18, 1930

SIDNEY D. WILGUS, M.D., *President, in the Chair*

A CASE REPORT OF A PATHOLOGIC SPECIMEN OF CHORIO-EPITHELIOMA MALIGNUM. DR. HALE A. HAVEN (by invitation).

This case is thought to be of interest for two reasons: (1) because the comparative rarity of the disease permits few men to observe more than one or two cases in a lifetime; many physicians, in fact, never see it at all; and (2) because of the definite clinical neurologic manifestations that are explained on the basis of the definite location of the intracranial lesions.

A white woman, aged 28, who had been married four and one-half years, entered the Passavant Hospital on Sept. 25, 1930, on the service of Dr. Arthur H. Curtis. She had had but one pregnancy. A hydatid mole had been removed two years previously by her family physician, and was reported histologically benign. She had had profuse bleeding prior to that curettage. She then had no flow for nine months and a scanty flow thereafter until five months before observation, when the menses ceased. She had been spitting up small amounts of bright red blood and small clots for five months. She had had diplopia for three weeks and had lost 16 pounds (7.3 Kg.) in four months.

Examination revealed an emaciated woman with pallid facies. There were diffuse, scattered areas of dulness over both lungs, with coarse râles audible on

auscultation. There was palsy of the external rectus of the right eye. A soft lipoma-like nodule was noticed beneath the skin in the region of the right sterno-clavicular articulation. Pelvic examination revealed the external genitalia to be normal, the cervix normal and the fundus of the uterus one and one-third times the normal size. The anterior outline of the fundus was somewhat irregular, not definitely nodular but with a tendency to decreased consistency. Hegar's sign was strongly positive. Roentgenograms of the chest showed the lungs to be riddled with tumorous masses similar to those observed in patients with sarcomatous metastases. The skull and the long bones were roentgenologically normal.

A diagnosis of chorio-epithelioma malignum was made and a fatal prognosis was given. The patient returned home and death occurred on Oct. 5, 1930. In the week preceding death, it was noted that there had developed a weakness of the right lower part of the face, of central origin, and that the patient complained bitterly of numbness and tingling paresthesias over the entire right side of the face.

An autopsy was performed about four hours after death. A primary tumor was found in the uterus with a multitude of metastases scattered throughout the abdominal viscera and lungs. The nodule palpable in the sternoclavicular region was a metastatic tumorous mass. A small nodule was noted near the right occipital pole of the brain. The gyri of the left hemisphere appeared to be flattened out, especially in the neghborhood of the rolandic area, and there was some discoloration about the region of the centers for the arms and face. The right fifth nerve was so soft that the slightest tension caused it to separate. The cause for this was investigated and another tumor nodule was found invading the ganglion and the paratrigeminal region on the right. This accounted for the symptoms referable to the fourth and fifth cranial nerves on the right. On section of the brain after hardening in formaldehyde, it was noted that there was a nodule just beneath the cortex in the region of the motor area on the left side to account for the right facial weakness. The other nodule extended to the surface in the fairly silent area of the right occipital region.

Microscopic examination of stained sections revealed the characteristic appearance of the atypical form of chorio-epithelioma as classified by Marchand. The Langhan's cells were of only moderate frequency. No large syncytial masses were encountered, but small syncytial plaques were moderately frequent. There was a diffuse invasion of the tissues by cells of the type of chorionic wandering cells, large mononuclear or polymorphonuclear, deeply staining cells, giving the impression of sarcomatous tissue. These cells showed a marked tendency to attack and erode the smaller blood vessels, and resultant extravasations of red blood cells and fibrin were much in evidence. No remote changes in the brain were noted, but in the areas immediately adjacent to the tumor nodules there was a surprisingly great glial reaction, with many giant astrocytes in evidence and an appearance almost as if encapsulation of the tumor mass was taking place rather than destruction of the tissue by the tumor which is characteristic of this type of lesion in other organs.

I have been unable to find any definite statistics as to the frequency of intracranial metastases in cases of chorio-epithelioma malignum.

DISCUSSION

DR. ARTHUR WEIL: The presentation made by Dr. Haven is interesting not only because metastases of a chorio-epithelioma in the brain are rare, but because this tumor presents a type of cancer that may heal spontaneously. The question arises whether the spontaneous breaking down of the chorio-epithelioma, which has been demonstrated, is due to a local defense reaction of the brain tissue, or to the production of specific protective ferments. Abderhalden called to attention some time ago the formation of such ferments against chorio-epithelium which invade the blood in pregnancy. If one considers the action of the brain against the tumor, it is interesting to note that a protective membrane was formed around the tumor, which separated islands of the tumor from the rest of the tissue. In

sections stained by Holzer's method it was interesting to note the formation of a coarse fibrous glial wall and of the extremely large astrocytes which I never have seen before in the defense reaction of the brain against metastatic tumors or in the encapsulation of anemic foci.

DR. G. B. HASSIN: Were there any changes in the ganglion cells or in the blood vessels, or any other parenchymatous changes, and if so how did they affect the meninges in this case?

DR. HALE A. HAVEN: The only apparent effect on the blood vessels observed in this case was an erosion of the vessel walls by the tumor. The blood elements participated, perhaps, to some extent as in a foreign-body reaction. The leptomeninges were somewhat thickened in an area close to the tumor mass in the occipital pole, but otherwise there was no notable meningeal reaction.

FAMILIAL DISEASE RELATED TO MERZBACHER-PELIZAEUS DISEASE. DR. PETER BASSOE.

CASE 1.—A boy, aged 10, of Polish descent, came to the Presbyterian Hospital a few weeks before presentation, chiefly on account of incontinence of urine and feces and difficulty in walking; these conditions had become troublesome a year previous to admission. He was normal at birth and walked at less than 1 year, but did not talk well until the age of 6. Mental decline and difficulty in speech had existed for at least two years.

Examination.—The patient was obviously mentally deficient; he could count only to 10. His speech was slow, scanning and indistinct, and he pulled the corners of the mouth far to the side during his laborious talking. There was no nystagmus. Vision and hearing were good. The disks were normal. He walked stiffly and unsteadily, largely on the toes, and tended to fall backward when standing. There was ataxia of both legs and arms and considerable weakness of the extensors of the left knee. Both legs were stiff; there was marked pes cavus and an equinovarus tendency. The great toes (more so the left) were habitually bent far backward, but on stimulation of the soles there was flexion at the phalangeal joints. Ankle clonus was present. The knee reflexes were increased; the abdominal and cremasteric reflexes were present; the arm reflexes were brisk. Pinching any part of either leg caused exaggerated extension of the left great toe, while pinching of the right calf or thigh caused flexion of the right great toe. The neck was short, the head long, and the occiput vertical. Roentgen examination of the head gave negative results. The sternum was short. The Wassermann reaction of the blood and of the spinal fluid was negative. The Ross-Jones and Lange tests of the fluid were negative; the protein measured 40 mg., and the sugar, 67.4 mg. per hundred cubic centimeters.

CASE 2.—A girl, aged 9, a sister of the preceding patient, had been under treatment at the Central Free Dispensary for attacks of petit mal. She talked slowly, with a stiff face like that of the brother, had moderate spasticity of the legs and was mentally backward. Pes cavus was present. The knee reflexes were increased; the abdominal reflexes were obtained on the left side only; there was no Babinski sign. Moderate ataxia of the hands with athetosis of the fingers was present.

Family History.—The father was well, but alcoholic and shiftless; he had been in jail for nonsupport. The mother had been epileptic since the age of 14. The eldest brother had a difficult birth, developed convulsions and involuntary evacuations and became unable to walk at the age of 9. For eight months, before death at the age of 11, he was unable to talk. Necropsy, by Dr. R. H. Jaffé, revealed a large porencephalic area, especially on the left side, with great destruction of white matter, and Dr. Jaffé connected this with the injury received at birth. Nevertheless, it is interesting that the patient should have been fairly normal up to the age of 9 years. A brother, aged 3, is, so far, normal. A paternal girl cousin, aged 11, was examined by Dr. Scheftel, who found her defective, with

intention tremor of the right hand, athetosis of the fingers, spastic legs and slight ankle clonus, but no Babinski sign. A brother of this girl, aged 16, appeared to be defective.

DISCUSSION

DR. L. J. POLLOCK: Is there any shortening of the achilles tendon?

DR. P. BASSOE: Yes, the patient tends to walk on the toes and has a distinct pes cavus. This condition is also present in an older brother, and the sister has the same tendency. When this boy stands, he has a tendency to fall backward. There is something of an athetoid condition. There is no nystagmus.

DR. L. J. POLLOCK: If there is shortening in the achilles tendon, it would probably explain in part some of the reflex peculiarity, particularly in the great toe.

MERZBACHER-PELIZAEUS DISEASE (FAMILIAL CENTROLOBAR SCLEROSIS). DR. YETTA SCHEFTEL (by invitation).

In two cases the chief factors which, in the absence of histologic study, justified their classification as Pelizaeus-Merzbacher disease were: (1) the hereditary influences; (2) the clinical observations; (3) the chronicity, and (4) the tendency to remission. Three members of this family, two males, were affected. The mother was well; the maternal aunt and her daughter had had chorea, and the eldest child of this aunt was paralyzed from birth. The predominance of living females through the three generations was noteworthy. Thus, the grandmother had three sisters, all of whom died at about the age of 76. Of the children of the first of these sisters, six died as babies; of those of the second sister, two boys died young; of those of the third, two boys died young. The mother of my patients had one brother and three sisters living. One brother died at 6 months, and the other was shot and died at 7 years.

The outstanding symptoms in my patients were disturbances in speech, incoordination and athetosis, intention tremor, spasticity, intellectual retardation and psychic peculiarities. An inconstant Babinski sign and ankle clonus had been elicited. There were no sphincter or sensory disturbances.

The onset was in infancy in the elder, and at about 6 years in the younger boy. In both cases remissions occurred. The chronicity and the relative mildness of the symptoms in my cases reminded one of the milder cases among Pelizaeus and Merzbacher's fourteen patients. The oldest of these patients, at the age of 28, showed choreiform movements and athetosis. He was able only to peel potatoes and do coarse sewing. Pelizaeus, too, noted remissions, an inconstant clonus and intermittent tremor among the patients.

The clinical observations pointed to a subcortical, extrapyramidal lesion of the cerebrum and the cerebellum.

FAMILIAL SPASTIC PARALYSIS. DR. HARRY A. PASKIND AND DR. THEODORE T. STONE.

Three brothers, aged 11, 9 and 8 years, respectively, were the three eldest children in a family of five; a brother, aged 2 years, and a sister, aged 9 months, were apparently normal. There was no history of spastic paralysis in the family. The paternal grandfather was nervous and had "fainting spells" between the ages of 60 and 70. A paternal aunt had an attack of manic-depressive psychosis at the age of 20. The father had "fainting spells" at frequent intervals between the ages of 10 and 15, but had none subsequently. The maternal grandfather and the mother were both "nervous."

The onset and the course of the condition were closely similar in the three brothers, and the results of examination were practically the same in all. Pregnancy and parturition were normal in all three births. As babies the patients were all unusually quiet and inactive, and showed a rigidity that rendered dressing

difficult. They walked at the ages of 7½, 5 and 3 years, respectively. None of them ever talked or understood words. The mental level in all three was that of idiocy. All of them had had epileptiform convulsions.

In all three, examination revealed a spastic paraplegia, with exaggerated deep reflexes and absent abdominal reflexes. One showed a bilateral Babinski sign and the other two showed unilateral plantar extension. The gait in all was of the "scissors" type; all showed genu valgum and flexion at the knees and hips; one also had a slight equinovarus deformity. The movements of the hands and arms were clumsy. Vision and hearing were present in all three, though it was not possible to determine the degree. There were no evident defects of the cranial nerves, no atrophy of muscles and no tremor, or choreic or athetoid disturbances of movement. The Wassermann reaction of the blood was negative in all three children and in the parents.

The eldest child died in June, 1930, of bronchopneumonia, and an autopsy was performed, the details of which will be reported later. Macroscopically, there were no changes in the cerebellum, pons, medulla or spinal cord. The cerebral dura was thickened and opaque, and densely adherent to the skull. The rolandic fissure was deeper than normal; there was complete absence of the sulci and convolutions in the parietal and occipital lobes, with an absence of blood vessels. Remains of sulci and convolutions were observed at the tips of the occipital poles, especially on the left, and one definite sulcus was observed on either side of the occipital poles. The medial aspects of the hemispheres showed many sulci and an almost normal appearance. The cut surface showed apparently normal gray matter with marked reduction of the white matter. The lateral ventricles, corpus callosum, basal ganglia and the third and fourth ventricles were apparently normal.

The literature of familial spastic paralysis was reviewed by Rhein (*J. Nerv. & Ment. Dis.* **44**:115 [Aug.] 1916). Adding to his figures the cases that have been recorded since his report, the disease has been observed in 148 families; 30 times in two generations, 6 times in three generations, 5 times in four generations and once in five and six generations, respectively.

DISCUSSION

DR. G. B. HASSIN: In spite of the enormous literature on cases of spastic type in general, cases of the kind demonstrated tonight are rare. In my seventeen years at the Cook County Hospital I have seen only one group.

The patients, a Polish girl, aged 22, and a boy, aged 17, were seen by me last June. Both were mentally defective and became sick at the age of 12. Up to that time they had been well. The first sign, according to the history, was pes cavus, just as in the patients of Dr. Scheftel and Dr. Bassoe. This was followed by difficulty in walking owing to the spasticity which was also present in the upper extremities. The spasticity was associated with marked Babinski and Rossolimo signs. In addition to clubfoot and spasticity, both patients had nystagmus, and in the girl, Dr. Suker diagnosed a secondary optic atrophy. The speech in both was slow, nasal and resembled a scanning speech; the abdominal reflexes were absent; the pupils were normal. The serologic observations were normal. One could not have clearer evidence for a diagnosis of multiple sclerosis, which was justified, especially in the girl, and which was diagnosed by Pelizaeus himself in his cases recorded in 1885 (a family type of multiple sclerosis). We thought that our cases might be Merzbacher-Pelizaeus disease, the more so because to our surprise the mother brought a third child, who was taken sick at the age of 14 with the same symptoms, including the optic changes the nature of which we could not determine. A fourth member of the family was said to have died of a similar condition. Altogether there were eight siblings, and of these, four were taken sick at about the same age and the disease in all instances ran approximately the same course. There were, in addition, marked mental disturbances; one patient had pronounced delusions and had to be placed in the

psychopathic hospital. The mother stated that her husband had been apparently well, as were also all uncles, aunts and cousins. For this reason I hesitated to diagnose the cases as Merzbacher-Pelizaeus disease, because that disturbance is hereditary, and is transmitted from males through females, who themselves are usually not affected. Only two women were known to suffer from this disease in the groups reported by Merzbacher and by Bostroem. In the absence of hereditary features, which were also absent in the cases of Bassoe and of Scheftel, the only diagnosis that could be made was that of a family or possibly a congenital spastic paraplegia. Probably the scant knowledge of this disease is due to the absence of extensive pathologic studies. These have been made only four times and the conclusions were that Merzbacher-Pelizaeus disease is degenerative, but differs, nevertheless, from multiple sclerosis.

Stone's contribution to the pathology of spinal spastic paraplegias, which may be syphilitic, is of great value, as histopathologic studies of such conditions are exceedingly scarce. Why a patient should have a normal pyramidal tract and have, as Dr. Paskind pointed out, a spastic paraplegia, is hard to tell. Rhein, in a publication in 1909 (*Am. J. M. Sc.*), explained such a seeming anomaly by the observation that pyramidal fibers are present but that they are so thin that they are incapable of transmitting the impulses. I would suggest that Dr. Stone make longitudinal sections to see whether the pyramidal fibers are as thin as suggested by Rhein.

DR. ARTHUR WEIL: I had an opportunity to study Dr. Stone's slides and saw that the islands of subcortical gray matter contained cells that showed all the characteristics of ganglion cells. I think that Dr. Stone's histologic diagnosis of "abiotrophy" should be changed into "heterotopia" of the gray matter. One should not apply Gower's idea of breaking down of already formed nerve tissue from lack of vital power to such a congenital displacement of the gray matter. There are instances published in which such heterotopias were combined with spastic paralysis and idiocy. Instances can be found in H. Vogt's monograph from the Zurich Institute and in earlier publications of Wohlwill on heterotopias. I think that one can indulge only in theories if one tries in such cases to localize anatomically the clinical manifestations.

DR. PETER BASSOE: This whole subject is complicated because, as Dr. Hassin said, it is largely based on clinical observations of overlapping conditions with great lack of anatomic control. Another difficulty is the differing nomenclature in different countries. I have not made any real study of the literature, but in the course of my observations I have tried to select a few facts that might help me in becoming oriented. In this mass it seems to me that I can see a certain type of disorder in which there are as the most striking lesions large degenerative or inflammatory foci in the white matter of the brain. This led Foix to use the term centrolobar sclerosis. He described acute and chronic forms. His acute forms correspond practically to Schilder's disease, in which, in addition to the extensive destruction of myelin, there are evidences of inflammation. Some of his chronic types correspond to Merzbacher-Pelizaeus disease. In a rough way, this may help one to become oriented.

I was a little disappointed that Dr. Stone did not show more large pictures of the myelin sheaths. I would like to have had him make sections with a Gudden microtome through the whole hemisphere. I think of this particularly because the last, which I believe to be the fourth, case examined anatomically and reported by Bodechtel (*Ztschr. f. d. ges. Neurol. u. Psychiat.* **121**:487, 1929), gave striking evidence of extensive demyelinization by this method. The myelinated fibers in the cortex and subcortex are comparatively well preserved, and the internal capsule and pons are clearly visible; but all of the white matter of the hemispheres is unstained, except for islands around the vessels, which gives a peculiar and characteristic appearance. The first picture that Dr. Stone showed looked much like the picture in this article, and that made me think that his case might belong in this group.

DR. THEODORE T. STONE: This is only a preliminary report. The brain is being studied in detail. The other half of the brain is being prepared to study with myelin sheath stains. According to the literature available, cases of this type have never been mentioned in American reports.

So far as Dr. Hassin's suggestion about the transverse and longitudinal sections of the spinal cord is concerned, I think that it is an excellent idea.

As to the pathology mentioned in a few of the histories that I gave tonight, there is no one who discusses the pathology who agrees with any other investigator regarding what he finds. There is no similarity in the pathologic observations in supposedly familial spastic paralysis. This can be explained because of its protean character. Various tracts may be involved and retrograde changes may be found. In our case, although we found definite changes in the ganglion cells, there were no disturbances in the tissues in or near the cortex, or in or near the vessels. We were unable to find any changes in the pyramidal tract system after it left the precentral gyrus. The brain was otherwise entirely normal; the centrum ovale was entirely normal, and it is difficult to say that we are dealing with a degenerative lesion without being able to find other changes that have been described previously as degenerative processes.

Regarding Dr. Bassoe's statement, I may say that large coronal sections of the brain are being studied now for changes in the myelin sheaths. I did not give this disease a name, and I purposely omitted the term heterotopia. In many instances this had been described only to be changed later, when the condition was found to be a defect caused by removing the brain or by some other trauma. Dr. Paul Schilder, when in Chicago, saw these slides and thought that the case was primary heterotopia.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 19, 1930

WILLIAM B. CADWALADER, M.D., *President, in the Chair*

A DEMONSTRATION OF WECHSELMANN LUMBAR PUNCTURE NEEDLES. DR. J. V. KLAUDER.

A presentation was made of three spinal needles, two of foreign make, the Wechselmann and the Dattner, and the third, the Doyle needle. These consisted of an outer needle which contained an inner, much finer needle. The outer needle acted as a guide and was inserted just short of the dura; the finer needle was then employed to puncture the dura. The caliber of the outer and inner needles of the Doyle needle was much larger than that of the other two needles and was otherwise different. Dr. Klauder did not prefer it to the foreign ones. The advantages of these needles concern the very small opening which it is possible to make in the dura; this prevents or minimizes subsequent leakage of spinal fluid, and in this way headache due to lumbar puncture is prevented or the incidence is greatly lessened. Such a needle is employed commonly in the German clinics on ambulant patients. A number of writers have reported favorably as to decreased incidence and severity of symptoms due to lumbar puncture (Eicke: Die ambulante Lumbal-Punktion mittels Capillarnadel in Theorie und Praxis, *Dermat. Ztschr.* **58**:57, 1930).

The employment of this type of needle is more difficult than that of the ordinary type; practice is required in order to use it successfully on all patients. The difficulty particularly concerns stopping the insertion of the outer needle just short of the dura. If the outer needle punctures the dura, of course the purpose of employing this type of needle is defeated. Another obstacle concerns the difficulty in obtaining much fluid, since the inner needle has such a small caliber; spinal fluid runs out very slowly; indeed, in some patients one is not certain that the

inner needle has punctured the dura until a syringe is attached to the opening and fluid is obtained by aspiration. The employment of a syringe in this manner overcomes the foregoing obstacle.

In patients who do not cooperate, whose muscles are held rigid and who are obese, the use of such a needle is particularly difficult.

A Case of Polycythemia Vera. Dr. William Duane, Jr.

Although there have been numerous case reports and studies of polycythemia vera, with nervous signs and symptoms which have often led to mistakes in diagnosis and in some instances to cerebral operations, this is the first case to come to attention in the neurosurgical section of the Hospital of the University of Pennsylvania. Christian, and more recently Brochbank, clearly pointed out the difficulty of making a correct diagnosis in the face of vascular accidents, particularly if the cyanosis or enlargement of the spleen is not marked.

N. R., a man, aged 24, a Jewish lawyer, was admitted to the neurosurgical section of the Hospital of the University of Pennsylvania, complaining of paralysis of the right hand and difficulty of speech. The history of the illness revealed that he had been perfectly well until Sept. 25, 1930, when, while driving his automobile, he suddenly became aware of a subjective stiffness and numbness of the right side of the body, which began in the right side of the face and spread down the neck, right arm and trunk to the upper third of the right thigh. This passed away shortly, but he had since had frequent similar attacks under the stress of mental strain or excitement. Two weeks after the onset of the illness, while writing, he suddenly had a sensation of numbness in the right hand. The fingers became paralyzed so that he was unable to grasp the pen or to extend the fingers. He was seen at this time by a physician who observed, besides the partial paralysis of the muscles of the right hand, some redness of the posterior pharyngeal wall, soft palate and tonsils, as well as enlargement of the tonsils.

From about this time on, the patient began to have slight occipital headaches and discomfort, but he continued actively to argue cases in the courts of law.

On November 20, when leaving a thrilling motion picture, he noticed for the first time that he had great difficulty in enunciating. He could not say the things he wished because it was hard for him to talk. He would stop and stammer, and his jaws became fatigued. There was no difficulty in swallowing, nor did food or water come back through his nose. Simultaneously, he observed that the right corner of the mouth drooped. On this and the following day he felt nauseated for the first time in six months, but did not vomit. His physician saw him again and observed a right facial weakness, central in origin, involving only the right corner of the mouth and right eyelid, in addition to difficulty in speech and persistent paralysis of the right hand which had not changed to any great degree.

It was thought that, owing to evidence of progression, this was a case of neoplasm of the left motor cortex in the region of the areas controlling the hands and face. He was therefore advised to go to the hospital to see Dr. Spiller and Dr. Frazier with a view to an exploratory decompression.

The patient's systemic, family and social histories were irrelevant, other than that he had been working under a severe strain for the past two years. From the past medical history it was learned that he had had measles as a child, but had never had any injury to the head, operations or other illnesses. He said that he had not had venereal infection.

The patient was young and mentally alert; he flushed readily on the least physical effort; his ears appeared somewhat cherry-red, and his lips showed a definite cyanosis, as did the beds of the finger-nails. There was a small, tender spot, the size of a twenty-five cent piece, in the left temporal region of the scalp. The tongue was heavily coated, and the tonsils were hypertrophied, cryptic and injected. The soft palate and pharynx were likewise injected. The lungs and heart were normal. The edge of the liver was barely palpable below the right costal margin, and the spleen was definitely palpable, firm and smooth, and on

deep inspiration it extended from 1½ to 2 inches (3.8 to 5 cm.) below the left costal margin. The blood pressure in both arms was 154 systolic and 100 diastolic. On neurologic examination, subjective disturbance of sensation was found, which the patient called a spasm or paresthesia, confined to the trigeminal distribution on the right side, but including also the right upper limb, the trunk and the upper third of the thigh. He had no motor or sensory aphasia, but a dysarthria. While the face was at rest there was no distinct difference in the muscles about the mouth on the two sides, but when he talked he innervated only the left side of the face; the right side was completely paralyzed. This paralysis was of the central type and involved the right orbicularis palpebrarum muscle to a slight degree. The masseter and temporal muscles contracted normally on each side. He was still able to whistle imperfectly, and he could blow out a match 1½ feet (45.72 cm.) away from his mouth.

The grasp of the right hand was fair, but not equal to that of the left. The individual movement of the fingers of the right hand was imperfect. He could not approximate the thumb to each finger separately. When he held the arms extended, the proximal phalanges of the right hand were overextended, and the middle and distal phalanges were partially flexed. When the right hand was overextended to the full degree, the flexion of the distal phalanges became more marked. There were no objective disturbances of sensation in either hand or the face. The biceps, triceps and radial reflexes were distinctly more prompt on the right, as were also the right patellar and achilles reflexes. There was a positive Hoffmann sign in the right hand, but no Babinski sign or ankle clonus.

An ocular examination gave entirely normal results, except for a slightly larger pupil in the left eye. There was no evidence of choking of the disks. The vessels appeared normal. The visual fields showed a very slight bitemporal cut, while the visual acuity was 6/7.5 in both eyes.

A lumbar puncture was performed, the initial pressure being 160 mm. of water. The fluid was clear and colorless and contained 4 red cells and 3 lymphocytes. The Wassermann reaction of the spinal fluid was negative, and the globulin, 1.8. Roentgen examination of the skull, heart and lungs gave negative results. The Wassermann reaction of the blood was negative. The basal metabolic rate was plus 12. Two urinalyses showed a specific gravity of 1.015 and 1.020, an acid reaction, very faint traces of albumin and from 2 to 4 leukocytes.

Under the supervision of Dr. Fitz-Hugh, studies of the blood were made. The viscosity was 8.6, the hematocrit reading was 53 per cent, and the capillaries of the nail beds showed the typical wide loops of the polycythemic type. On three examinations within two weeks, the red blood cell count varied from 6,500,000 to 7,000,000; the white cells from 8,800 to 6,700, and the hemoglobin from 135 to 142 per cent. Two differential counts showed: neutrophils, 90; lymphocytes, 14, and large mononuclears, 5 per cent on one occasion, and neutrophils, 73; lymphocytes, 19; large mononuclears, 3, and eosinophils, 5 per cent on another. The platelets numbered 350,000, and reticulated red blood cells 1 per cent in a wet preparation. The fragility began at 0.450 and was complete at 0.350. The venous coagulation time was twenty-nine and twenty-one minutes.

From the history of a sudden onset of the symptoms, usually following excitement or effort of some sort, from the physical conditions of cyanosis and an enlarged spleen in conjunction with the increase in the number of blood cells and the other observations of the blood, Dr. Spiller, Dr. Frazier and Dr. Fitz-Hugh reached the conclusion that the patient's cerebral signs and symptoms were due to a vascular accident incident to polycythemia. An operation was thought inadvisable for this reason.

The administration of phenylhydrazine hydrochloride, 0.1 grain (0.006 Gm.) three times a day for twenty doses, was begun. At this time the blood contained 80 per cent hemoglobin and 4,200,000 red cells; the blood pressure was 130 systolic and 60 diastolic. The patient felt comfortable, except for the residual paralysis already mentioned, namely, a weakness of the right side of the face and the right hand. He was discharged on December 15, showing possibly a slight improvement in the dysarthria and paralysis.

DISCUSSION

DR. N. W. WINKELMAN: I am disappointed in not hearing Dr. Spiller discuss this case. I have recently had the opportunity of studying two cases of polycythemia vera, in one of which I obtained an autopsy. This is the only case of the kind that I have had occasion to examine. In this case the outstanding feature was the tremendous dilatation of all the blood vessels, particularly the veins, throughout the entire central nervous system. No actual hemorrhages were discovered, but one could easily imagine that they could occur as the result of the intense dilatation. The cerebral tissues suffered damage only as a consequence of the enlarged vessels, in the same manner that one could expect from any other expanding small lesion. The other case was a clinical one. Mental symptoms predominated, and the patient was sent to a nearby institution.

DR. T. B. HOLLOWAY: I think that I understood Dr. Duane to say that the eyes of his patient were normal. In 1912, I reported the ocular observations in certain conditions associated with cyanosis. As is well known, in polycythemia there are changes in the caliber of the vessels and in the color of the blood stream. Changes in the disks are also present. These observations are practically duplicated in certain cases of congenital heart disease. A third group constitutes those due to poisoning by drugs, notably by the products of coal tar.

I think that Dr. Stengel was the first to point out the possibility of confusing polycythemia with chronic poisoning caused by acetanilid.

PECULIAR SYMPTOMS OF THE ARM AND HEAD IN CHRONIC EPIDEMIC ENCEPHALITIS. DR. A. M. ORNSTEEN.

This article will be published in full elsewhere.

TRAUMATIC EPILEPSY TREATED BY EXCISION OF THE CEREBRAL CORTEX ACCORDING TO THE PENFIELD-FOERSTER METHOD. DR. REED HARROW.

Dr. Harrow said that he believed this report would be of interest, since it was the first case in the department of neurosurgery of the University of Pennsylvania Hospital in which the patient was treated by radical excision of the cerebral cortex for posttraumatic epilepsy, as advocated by Penfield and Foerster.

R. E., a boy, aged 18, was transferred from the service of Dr. William G. Spiller to the neurosurgical department, service of Dr. Charles H. Frazier, University of Pennsylvania Hospital, On Oct. 22, 1930, complaining of convulsions. The patient had been well until Nov. 19, 1927, when he received a gunshot wound in the left parietal region. The shot stripped a small area of scalp away from the underlying bone, but according to the statement of the attending physician, the skull was not injured. Immediately following the accident, there was marked weakness of the right upper extremity and some difficulty in motor speech. However, the wound healed rapidly, and the symptoms gradually disappeared. It was not until Easter, 1929, that he had the first convulsion. He was found in bed with the arms flexed, the legs and body rigid, and the eyes rolled upward; the tongue had been bitten. The attack lasted for several minutes and was followed by unconsciousness, which lasted three hours. Since this time he had had from six to eight convulsions every week, none, however, as severe as the first one. Phenobarbital had been administered with some benefit. Before an attack he would complain of a feeling of numbness, beginning in the right hand and forearm and extending to the right shoulder, loss of power in the right hand and forearm, soreness in the left parietal region, spots before the eyes and a singing in the ears.

The previous medical and family histories were essentially unimportant, aside from the fact that two maternal aunts were said to have had migraine. There was no history of epilepsy.

The patient was well developed and robust, without gross abnormalities, except the scar in the left parietal region in the same vertical plane as the ear and just below the sagittal suture.

Neurologic examination showed the patient to be shy and reserved, with a fair degree of intelligence and slight hesitancy in speech. Although the patient was right-handed, the grip of the right hand was considerably weaker than that of the left, the dynamometer readings being 135 and 160, respectively. Motor power elsewhere appeared normal. The patellar and achilles reflexes were slightly hyperactive. Objective sensory disturbances were not present. The cranial nerves were normal. Observations of the epileptic seizures showed that each attack began in the right upper limb. The head was turned to the right, the eyes to the left. The convulsive movements were at first tonic, then clonic, involving all of the limbs, but were especially pronounced on the right side. The attack usually lasted from two to three minutes, consciousness soon being regained.

The Wassermann test of the blood and other laboratory tests gave negative results.

Roentgenograms of the head showed that the pituitary fossa was within normal limits — anteroposterior, 10 mm., and depth, 8 mm. Evidence of a pathologic process in the bone was not found. The report from an encephalogram, made on Oct. 18, 1930, was: probable subcortical lesion with arachnoiditis on the left side, deforming the lateral ventricle. The subarachnoid markings on the right side appeared normal; on the left side, the markings were not as prominent as those on the right. There was a slight deformation of the left lateral ventricle, suggesting that there had been a subcortical, or rather a cortical, injury to the brain which had caused displacement of the roof of the ventricle toward the lesion. This was present in the parietal region.

After careful consideration of the observations that pointed to a cortical scar, serving as an epileptogenic focus in this case, it was decided that cortical excision should be attempted. On November 10, Dr. Frazier operated under local anesthesia. A bone flap was reflected to uncover the motor cortex and that region of the cortex underlying the cicatrix of the scalp which represented the site of the gunshot wound. In making the dural incision, it became manifest that in the upper and posterior quadrant of the opening the dura was adherent to the cortex for an area representing approximately the diameter of a silver dollar. This area, including the dura, was removed for a depth of from 1 to 1.5 cm. by means of the Bovie knife. When the electric knife was first applied to the cortex, at the site of the scar, a typical epileptic seizure was precipitated. A second convulsion occurred before the scar was entirely removed, and in order to arrest further attacks, the patient was given ether. The dural defect was replaced by a strip of fascia lata obtained from the thigh. After careful hemostasis, the wound was closed. Dr. Frazier was of the opinion that the greatest part of the cortical scar was just posterior to the rolandic fissure.

Following the operation, the patient was given 500 cc. of citrated blood. His condition was satisfactory. When he regained consciousness, it was noted that there was considerable weakness of the fingers of the right hand and the right corner of the mouth, but he was able to move the right arm and forearm in all directions. The next day it was observed that there was slight difficulty in motor speech. That night he inadvertently fell out of bed, but did not appear to suffer from ill effects. On the second postoperative day, the right hand and forearm were very weak, and the impairment of speech became more pronounced. At 2:30 a. m., the respirations dropped to 10, with complete paralysis of the right upper extremity and motor aphasia, and the patient was unable to protrude the tongue. Muscular twitchings were noted involving the right side of the face and right upper extremity. Sixty cubic centimeters of 50 per cent dextrose was administered intravenously; a spinal puncture revealed the fluid to be clear and colorless and under normal pressure. Throughout the night and the next day there were numerous attacks of clonic contractures of the right facial muscles and of the right arm and forearm. Dextrose and phenobarbital administered intravenously had practically no effect in controlling these convulsions. At no time, however, did the convulsions become generalized.

On the fourth postoperative day, the clonic movements of the right upper extremity and right side of the face became less frequent and within a day or so completely disappeared. The patient was now able to move the right arm and forearm, he could move the hand and fingers to a less degree; he was able to say "yes" and "no" in response to questions; the tongue could be protruded, and general improvement was noted.

On December 1, a complete neurologic examination was made, and it was found that the grip of the right hand was 40 compared to 110 of the left; the right forearm was slightly weaker than the left; fine movements could not be executed with the right hand and fingers; there was complete astereognosis and loss of sense of position in the right hand and fingers. The right patellar and achilles reflexes were exaggerated, and an abortive ankle clonus was obtained. Noticeable weakness was not observed in the right lower extremity. The right facial weakness, central in origin, persisted. There was a marked motor aphasia, and the patient had slight difficulty in reading written words.

The patient was then transferred to the service of Dr. Spiller. An examination made on December 10, showed considerable improvement. The grip of the right hand was 65, and that of the left 110; a detectable weakness was not found in the right forearm; the position of the thumb and fingers of the right hand was recognized about 60 per cent of the time; astereognosis was still present; difficulty was experienced in moving the fingers in the usual manner to recognize an object placed in the hand; speech had improved 50 per cent, the patient now being able to speak short sentences in a slow and somewhat hesitant manner; he read an advertisement and explained its content. The facial weakness was still present, and the patellar and achilles reflexes were slightly exaggerated. Ankle clonus was not obtained.

The patient was not seen again until December 15. At this time the grip in the right hand had increased to 85, while that of the left was 115; 3 pound (1.4 Kg.) weights could be held in any position without difficulty. Stereognostic sense and sense of position were normal; power of speech had improved markedly, the patient being able to speak almost as well as before the operation; facial weakness was still present; the right patellar and achilles reflexes were slightly hyperactive; all other reflexes were equal and normal. The examination otherwise gave negative results.

At no time since the operation, a period of one month, had there been any convulsive seizures. The patient had not complained and was much pleased with the results.

An examination of the specimen removed at the operation was made by Dr. Alpers. His report was as follows: The section consisted of a piece of dura and cortex. The dura was slightly thickened and showed an increase in cells. In some areas this was focal, the cells being fibroblastic. The pia-arachnoid was somewhat thickened and vascular, and was adherent to the cortex. The dura, though in gross it appeared adherent, was much less so than one would have surmised; however, it was adherent. The upper layers of the cortex in some areas and the entire width of the cortex in others were the seat of rather marked changes. The most common change was a marked mesodermal reaction in the cortex with the presence of dense bands of collagen fibrils and vascular spaces. These had apparently come from the pia-arachnoid, which could be seen dipping down into the cortex in places. Fibroglia fibrils were numerous among the collagen bundles. The remains of cortical cells were seen everywhere; the scar was more fibrous than vascular. The deeper cortical layers were very much rarefied, as compared with the dense fibrous upper layers. In one area of the cortex a wedge-shaped area of gliosis constituted the cortical reaction, rather than the mesodermal invasion previously mentioned. In the latter there was surprisingly little glial response. In the white substance below the cortex the astrocytes were hypertrophied and possibly somewhat hyperplastic — there seemed to be a formation of new vessels in these areas and also marked congestion. The vessels often showed adventitial proliferation. Phosphotungstic stains showed a gliosis in the white matter.

DISCUSSION

DR. W. B. SPILLER: The patient, as a result of a gunshot wound in the left parietal region, had frequent, severe, convulsive jacksonian attacks which began in the right hand. At my request Dr. Harrow made an encephalogram. This showed a displacement of the left lateral ventricle toward the left, the site of the trauma. The case seemed suitable for the Foerster-Penfield operation. I explained the German text of the paper by these two authors to the patient's elder brother, showed him the diagrams of the lesions and read to him the reported results of the operations. The patient and his family insisted that they wished the operation. I then requested Dr. Frazier to perform it. During the first few days following the operation, the patient's condition was serious, but within a week or ten days he began to improve, and at present he seems to be in almost the same condition as before the operation, but he has had no convulsions for about four weeks.

Collier, in an important paper on recent views regarding cerebral localization, mentioned that in the chimpanzee's brain as determined by electric stimulation each motor cortex may be excised without causing permanent paralysis, but no motion can be obtained from such a brain by electric stimulation. He has little to say regarding the human brain in relation to paralysis produced by excision of a part of the motor cortex, although he refers to the well known case of Horsley.

The case reported is one in which the excision seemed to me to extend into the motor cortex, although Dr. Frazier is doubtful of this. It is difficult to determine the exact limits of a cortical lesion in the exposure of the brain made at operation. This case and other cases in which the Foerster-Penfield operation has been performed should advance our knowledge of cortical localization.

DR. C. H. FRAZIER: Dr. Spiller has covered the most important aspects of this problem, and we all remember, on the occasion of Dr. Penfield's visit a few months ago, his recital of his own experiences with the operation in question.

With regard to the precise site of the cicatrix in the case under discussion, there may always be a reasonable doubt as to the identification of the rolandic fissure, but I felt confident that the scar was posterior to the fissure. The character of the patient's aura (a sense of numbness in the arm) and the loss of stereognostic perception might be accepted as reasonable evidence that the lesion was posterior rather than anterior to the fissure.

Two points in the technic might be discussed. How far below the surface should the resection be made, and by what method should the tissue be resected? As to the former, I presume that one must be governed by the appearance of the site to the naked eye. I used the electric knife in the removal of the lesion. Penfield makes his section of the tissue with a suture mounted on a curved needle. As the suture is tied, the tissue enclosed is divided. I am disposed to believe that this method should be preferred, as it inflicts less damage on the tissues.

DR. T. S. FAY: Dr. Harrow has presented an interesting and most important case. It affords a new means of approach to the subject of the convulsive state. I believe that Dr. Frazier noted that the patient had a convulsion while he was excising the scar with the electric cautery, and that another convulsion followed the removal of the motor area. It is evident, if we accept the dictum of Hughlings Jackson, that a diseased or damaged motor cell cannot give rise to increased activity, and as this patient had a convulsion during and after the removal of the tissue of the brain, there must have been active motor cells that were not included in the area removed. This would further substantiate Dr. Frazier's belief that he was operating behind the true motor area and not exactly in it. The astereognosis and sensory aura would also indicate that the lesion lay behind the rolandic fissure. The facts that on the fourth day following the operation the paralysis was more profound and that gradual improvement has been noticed to the present time clearly indicate to me that the true motor area was not removed and that probably cerebral edema of the adjacent areas may account for the variability of the paralysis.

It has seemed to me that a scar within the substance of the brain cannot of itself produce a convulsive seizure, for if this were so one would expect a constant influence from such a fixed lesion and either status epilepticus or epilepsia partialis continuens. No matter what importance one attaches to the scar, one must admit some additional cyclic and recurrent factor, which comes and goes, to permit the patient intervals when he is entirely free from attacks as well as from subjective or objective signs of the cerebral lesion.

I believe that Dr. Frazier's point is well taken, that the adherence of the scar in the brain to the dura is a most important factor, but I would emphasize the fact that the arachnoid and fluid pathways are also caught between the lesions and form a most probable obstruction to the cerebrospinal fluid. Dr. Frazier has removed many tumors from this area that have necessitated leaving scars within the brain, but convulsive seizures have not always followed these operations. He is most careful to protect the brain from the dura and to repair the dura after all bleeding has been controlled, so that such adhesions may be prevented.

I am strongly opposed to destruction of the tissue of the brain or removal of the motor cells to prevent convulsive seizures. These patients have lost too much tissue as it is, without taking more away. I do not believe that one is solving the problem by merely stopping the seizures. It is not the convulsion that one is so much concerned about; it is the progressive mental deterioration that goes with it. To remove tissue of the brain and produce a paralysis to stop a seizure will probably not stop the mental deficiency, even if the convulsions do not return. Only five weeks have passed since the operation in this case; I feel that five years would give a better estimate of the value of this procedure.

The Penfield-Foerster operation has been modified so as to dissect out only the scar of the dura and the arachnoid, leaving the cells of the brain intact, and satisfactory results have been obtained. Great care has been taken to free adhesions of the subarachnoid space; this can be done with a magnfying glass and a pair of fine arterial scissors. I believe that if one can preserve these cortical cells and at the same time free the adhesions from the dura and the arachnoid, one may expect even better results than those reported from excision of the motor area.

DR. B. J. ALPERS: The pathology of this scar was typical of that which Penfield described in operations in cases of epilepsy. He pointed out the extreme importance of removing the entire scar if a recurrence would be avoided. In the two or three cases in which a renewal of the epileptic attacks followed the operation, the entire scar had not been removed.

ATROPHY OF THE LIMBS AS A SIGN IN INVOLVEMENT OF THE PARIETAL LOBE. DR. ALEXANDER SILVERSTEIN.

For the past three years atrophy occurring in the muscles of paralyzed extremities due to cerebral lesions has been considered as significant of involvement of the parietal lobe. For this report Dr. Silverstein selected eight cases from a larger series of tumors of the brain which were verified at operation or post mortem. Comparative studies were made on hemiplegic patients without atrophy, who showed no clinical evidence of involvement of the sensory cortex. The localizing value of the atrophy was especially useful in patients with a clouded sensorium. Of the eight cases, five were tumors of the parietal lobe, one of which extended slightly posteriorly to the occipital lobe; another was in the parieto-occipital lobe; however, the manifestations in the occipital lobe of a right homonymous hemianopia appeared suddenly when the patient's condition was far advanced. One was in the parietotemporal-occipital lobe. One patient presented a glioma reaching from the tip of the frontal lobe on the right, including the temporal and occipital lobes without invading the parietal lobe; however, the white matter in this region was greatly increased, swollen and edematous. It is interesting and important to note that the latter, which was an end-result, explained the atrophy of the left hand that appeared for the first time tweny-four hours before the death of the patient. Of this series, four were endotheliomas, two of which had a more or less sudden onset and a rapid course, simulating a glioma.

Two were gliomas, one a hemangioma and one case, in which a diagnosis of tuberculoma had been made prior to operation by Dr. Winkelman, proved to be two such tumors which were removed with the electric cautery by Dr. Fay. The youngest patient was 19 years of age, and the oldest patients were 60 and 67, respectively, both of whom had encapsulated tumors which were removed by Dr. Fay.

All of the patients had atrophy of varying degrees, always in the paralyzed upper extremity, which first made its appearance in the interossei muscles on the dorsum of the hand, soon involving the forearm, arm and shoulder. The skin in these areas was smooth, glossy, moist and warm as compared with that of the opposite side. The atrophy could be easily discovered by palpation; it assumed the types described by Dejerine as those simulating chronic progressive muscular atrophy, and the main-en-griffe. As a rule, the atrophy of the muscles was unilateral, but in a few instances slight atrophy could be detected on the sound side late in the disease, without, however, any signs in the pyramidal tract. In a number of cases the progressive increase in the atrophy was also found to be in direct relationship with the increased intracerebral pathologic changes. In every patient associated sensory disturbance was found. Astereognosis, graphesthesia and sense of position and passive motion of the fingers and toes were lost in the affected extremities, whereas the sensations of pain and vibration were little involved. Occasionally, the atrophy of the muscles made its appearance prior to any marked sensory disturbance. It was also noted that with the advanced atrophy, the extremity tended to become flaccid, and the reflexes which were previously exaggerated seemed to disappear.

The first case to come under the observation of Dr. Silverstein was that of a girl, aged 19 years, who in November, 1927, gave a history of progressive dizziness, especially when changing her position, as in stooping or arising from a recumbent position. She complained of headaches, vomiting, visual disturbances and paralysis of the right side of the body. All of these symptoms dated back two and one-half years and were progressive. On admission to the Samaritan Hospital, under the care of Dr. Bockroch, she was semistuporous and blind in the right eye, owing to secondary optic atrophy, with 5 diopters of choked disk in the left eye. She had a complete right hemiplegia, with the atrophy so intense in the right upper extremity, with flaccidity, that a lower motor neuron form of paralysis was suggested. She had complete astereognosis in the right hand. Dr. W. W. Babcock removed a large encapsulated tumor about the size of an orange from the parietal area. The patient made a remarkable recovery as to function of the right side, and the atrophy practically disappeared within four weeks. Incidentally, she had been working steadily since that time. It was this case that first brought Dr. Silverstein's attention to the significance of atrophy of cerebral origin.

In two other cases, the course and clinical picture were typical of cerebral thrombosis, with normal eyegrounds and a pressure of the spinal fluid not above 12 mm. of mercury. In each case the progressive increase in the atrophy of the upper extremity helped to determine the presence of a lesion involving the parietal lobe. The day before the meeting Dr. Fay had removed an endothelioma in one of the cases — that of a patient 67 years of age.

In cases of hemiplegia, atrophy of the muscles, although repeatedly observed, has been imperfectly recognized and is a symptom which as a rule is often overlooked. In 1842, Hall wrote of it, and many later authors, especially French and German, have discussed the subject. Many theories have been advanced as to the pathogenesis of the atrophy. Arthritic changes, the cerebral cortex, especially the optic thalamus, the centrum ovale and vasomotor disturbances have been offered as explanations for the phenomenon. Beginning with Charcot, Brissaud and many others, and more recently Schaffer, Margulies and Kiss, the anterior horn cells of the spinal cord have been considered as the cause. Some authors have reported reaction of degeneration in the atrophied muscles; however, Dejerine and many other writers have not found these changes in the anterior horn cells.

The two cases reported by Brissaud, in addition to softening in the prerolandic area, had involvement of the postrolandic and parietal areas. In reviewing the cases of a number of other writers, it seems that the parietal lobe was involved directly or indirectly in the pathologic process. Of American writers, J. H. Rhein, in 1927, reported eight cases of central atrophy. He reported his first case to this Society in March, 1916. The patient was a man, 24 years of age, who received a gunshot wound in the parietomotor area with a resultant left hemiplegia and astereognosis. The atrophy was marked and of an unusual character. Rhein did not mention any sensory changes in his other cases. Dr. Spiller has clearly discussed this subject in the September, 1930, issue of *Progressive Medicine*, especially as to a German article by Joseph Kiss in 1929. The latter reported the case of a patient whose brain showed a hemorrhage into an angioma at autopsy. The author emphasized the rarity of this lesion. His sections revealed a tumor not only in the precentral area, but definitely in the parietal area. Time did not permit the detailed discussion of the contents of the article by Kiss. The author accepted the theory of Schaffer in considering the atrophy of the muscles as due to a disturbance in the peripheral neuron. Normally, the impulses passing from the motor cortex produce a biochemical equilibrium in the cells of the anterior horn in the spinal cord, whereas a lesion of the pyramidal tract produces a disturbance and abolishes this equilibrium and thus accounts for the atrophy.

Through the courtesy of Dr. Winkelman, Dr. Silverstein was able to show a lesion of the same type as that described by Kiss, but involving almost entirely the parietal lobe. It is interesting to note that in this case of a patient, 76 years of age, the resident physician at the Philadelphia General Hospital was able to describe an atrophy of the right side and astereognosis of the right hand. The case was reported by Dr. Waggoner in 1927.

It was necessary only to mention some of the points against the theory of Kiss, which are as follows: 1. All cases in which there is disturbance of the pyramidal tract should show this atrophy. This statement is not true, as some of the most intense disturbances in disease of the upper motor neurons, whether pure hemiplegias without the sensory cortex being involved or cases of posterolateral sclerosis with a marked spastic paraplegia and involvement of deep sensibility, do not show this atrophy. 2. The vasomotor signs that accompany the atrophy are in the form of smooth, glossy feeling to the skin and at times sweating. 3. Langworthy and Richter have described sweating by stimulating certain areas of the brain close to the motor cortex with the faradic current. 4. Sweating on the side opposite to cerebral lesions has been known to occur in infiltrating tumors. 5. Fay and Winkelman have both noted marked vasomotor changes, such as swelling and edema of the extremities, opposite a cerebral lesion involving the parietal area.

It can be said in summary that: 1. There is evidence that a trophic center is present in the cerebral cortex which sends its impulses to the lower centers in the same manner as do the various other systems. 2. Cerebral lesions in the parietal lobe give rise to atrophy in the paralyzed muscles. 3. The progression of the atrophy in some cases of tumor of the brain may be an index to intracerebral pathology. 4. The sudden onset of a hemiplegia with astereognosis and atrophy of the muscles should cause one to suspect a hemorrhage into a tumor of the brain.

DISCUSSION

DR. W. B. SPILLER: I have been interested in cerebral atrophy for a long time. Probably my attention was seriously attracted to the subject by a remark made to me by Dejerine in 1895, to the effect that in cases of hemiplegia he had seen this atrophy develop within six weeks and that it could not be attributed to disuse. Various explanations have been given for it. I know of no case in which cerebral atrophy has development as a result of a lesion confined to the parietal lobe. If the parietal lobe has an influence in causation, it must be in association with hemiplegia produced by a lesion of the pyramidal tract.

Dr. T. S. Fay: This subject has interested me for the past six years; in fact, ever since Dr. Spiller called my attention to the marked edema that characterized some cases of hemiplegia on his service at the Philadelphia General Hospital. In going over a number of these cases, I was struck by the proportion of left hemiplegias that showed the usual spasticity and atrophy. Dr. Mills has ascribed to the right temporoparietal area of the cortex the function of emotional augmentation and vasomotor properties that mirror the development of the left auditory areas of speech.

Henry Head described a type of cortical atrophy that he believed to be due to destruction of the sensory areas in the parietal lobe. The loss of values for sensory tone to the associated motor cortical cells gave rise to the atrophy in the limb in the same way that destruction of the posterior roots of the spinal nerves give rise to peripheral atrophy. In objective sensory tests, tabes is frequently associated with this type of atrophy when the posterior spinal roots are involved, although in muscle tests or electric reactions the motor elements of the anterior roots do not show involvement.

The type of atrophy that one sees when lesions involve the parietal lobe and later extend to the prerolandic zone is quite different from that of a primary lesion of the motor area or of the pyramidal system. The spasticity is greater in the latter case, the pathologic reflexes are more pronounced, and the paralysis is more marked.

I believe that Dr. Silverstein has called attention to a most important sign that has proved to be of localizing value in distinguishing tumors of the parietal lobe from those situated anteriorly in the frontal lobes.

Dr. N. W. Winkelman: Atrophy as the result of a cerebral lesion is not common. My ideas concerning this agree in all respects with the views expressed by Dr. Spiller. There are many cases of hemiplegia without evidence of atrophy, in the beginning at least. It has been my experience that when atrophy does occur early symptoms are usually present that one associates with involvement of the parietal lobe. In my experience I have seen but two cases in which symptoms were present in the parietal lobe alone. These occurred many years ago, and I do not recall whether or not atrophy was present. I believe that when early atrophy is present one can make a diagnosis of involvement of the parietal lobe in conjunction with the motor area. This rule has been of inestimable value to me in localizing lesions for my surgical colleague, Dr. Fay. Only yesterday, Dr. Fay removed a tumor from the parietal motor area which was correctly localized on the basis of severe motor symptoms plus atrophy, combined with the ordinary signs that one associates with involvement of the parietal lobe. Many times one cannot obtain sensory phenomena from patients because of stupor, and the atrophy then becomes a highly important sign.

Dr. A. Silverstein: I realized when I presented this report that it was equivalent to attempting an explanation of the pathogenesis of the Argyll Robertson pupil. We noted these cases clinically, and the investigation was suggested by the discovery at operation in the first case that the tumor was located posteriorly in the parietal area. Tumors in this region can easily press downward and produce hemiplegia. The paralysis is apt to be complete, with flaccidity. I do not believe that the problem is solved; this is only a clinical report based on cases that were verified at operation and post mortem.

Perimetric Examinations. Dr. Hazel Wentworth.

The basic principle and method of making perimetric examinations were described. Two types of examination were mentioned, the outlining of the field of vision by means of the perimeter and the minute search of the visual area for scotomas, in which a flat continuous surface or tangent screen was used. Various types of instruments were shown and their relative advantages and disadvantages pointed out. Particular attention was given to the Feree-Rand perimeter, which provides for the control and standardization of illumination, the size, color and

intensity of the test object and the background and similar nonpathologic factors which cause variation in the size and shape of the visual field. The sensitivity of the perimetric examination as a test of visual function was shown to be due to the magnification of the retinal image, the size of which increases as the square of the distance to which it is projected. For instance, in the case of a 1 mm. test object used at 33 cm., 1 meter or 2 meters was shown to be equivalent to a magnification of 22, 66 and 133 diameters, respectively, with a corresponding magnification of the area of 484, 4,444 and 17,777. In each case the retinal image was given as 0.045, 0.015 and 0.0075 mm. in diameter. The minuteness of the examination was thus shown to be limited only by the threshold of perception, which in the central field for a person with vision of 20/20 was given as 0.00001 mm.

Reference was made to the subjective difficulties met with in obtaining reliable results, such as the difficulty of obtaining adequate responses from ill, drowsy, aphasic or unintelligent patients, and the corresponding demands on the intuition and ingenuity of the examiner in obtaining cooperation and in interpreting responses.

The reliability of the results was shown to depend in large measure, however, on external factors within the control of the examiner. For instance, changes in intensity of the illumination used were shown to affect the size and shape of the visual fields: (1) through their effect on the sensitivity of the retina to color, which was shown to vary enormously with the illumination to which the eye was adapted, and (2) their effect on the intensity of the stimuli by the pigment papers generally used. As these are seen by reflected light, their intensity is directly dependent on the amount of light falling on them. Changes in the limits of the visual field of from 11 to 37 degrees, according to the meridian and color of the test object used, were shown to occur simply as a result of the changes in the illumination of the perimeter arm equivalent to the changes in daylight occurring during a single bright winter's afternoon. Changes in the limits of the field for color from a few degrees from the center of the field to the full extent of the field were shown as the result of changes in intensity of the stimulus alone. It was also shown that the limits of the fields for red, blue and green stimuli could be made to interlace or to be concentric in any order at will simply through changes in the relative intensity of the stimuli used.

Changes in the size of the stimulus were shown to have an effect similar to those of intensity on the limits of the field for color. Background was also mentioned as a variable factor. The blind spots for color were shown to be from 5 to 18 per cent larger when determined on black, and from 22 to 60 per cent larger when determined on white, backgrounds than when a background of gray of the brightness of the color was used. Similar variations in the size of the fields of color with different backgrounds were also demonstrated. It was finally pointed out that unless these and other variable factors external to the patient were controlled and standardized, as is the case when the Ferree-Rand perimeter is specified, any but gross changes in the fields were susceptible of a number of interpretations and their reliability as indicative of changes due to pathologic processes was obviously questionable. The illustrations were taken from the research studies of Dr. Wentworth and those of Ferree and Rand.

DISCUSSION

DR. T. B. HOLLOWAY: I wish to point out that during the past fifteen or twenty years just as many improvements have occurred in diagnostic methods in various branches of medicine and surgery as in many refinements in perimetry. At the University Hospital, the examinations of all visual fields are made in a room which isolates the patient from disturbing surroundings; they are made by the same examiner with a constant amount of light and color saturation of the test object.

It is well known how frequently borderline cases remain undiagnosed even after examination by various clinicians. Sometimes in these cases, an examination of the visual field offers the greatest possibility for a suggestive lead. Under these

circumstances the results of one examination should not be accepted; others should be made, for in basal conditions, especially, changes in the visual fields frequently occur within a few days.

The first question that I ask when I see the results of an examination of a visual field concerns the examiner. Just as some neurologists are not particularly clever in functional cases, just so certain ophthalmologists vary in their work on visual fields. It has always been remarkable to me that a patient racked with headache, nausea and even vomiting could be made actively and accurately interested in the movements of a small piece of colored paper. The fact remains that a clever examiner can obtain good results under such conditions; however, as in the examination of other organs, it must be remembered that the patient and not simply the eye is being examined.

In the moving picture of perimetric fields about to be shown, we have taken a definite number of actual fields and have interpolated others. Thus the first picture, which concerns a case of disease of the pituitary body, is made up of 33 actual fields and 207 interpolated fields.

MOVING PICTURES OF VISUAL FIELDS. DR. T. B. HOLLOWAY.

Moving pictures of visual fields were shown, and the cases were explained as they appeared on the screen.

DISCUSSION

DR. T. S. FAY: This is a remarkable demonstration of the type of analysis that is offered by moving pictures. I have been particularly impressed by the variations that the fields show over a period of time. There is a constant waxing and waning of the scotomas, a widening and contracting of the margins of the fields, and in cases of pituitary tumors the advance and retreat of the hemianopic field before the final closing-in process is complete suggests some variable in the intracranial pressure or visual acuity from time to time. Dr. Wentworth has already shown that every detail of the examination has been standardized, and with the examination of the visual fields made under identical conditions by the same examiner it is probable that the variable lies within the central nervous system.

It seems to me that considerable light might be thrown on these variations if the patient were placed on a fixed intake of fluid, so that intracranial pressure would be more or less fixed from time to time, and a reading of the blood pressure made at the time of each examination of the visual fields. From the standpoint of intracranial pressure, the two most likely variables within the closed confines of the skull are circulating blood and cerebrospinal fluid. As we have been able to maintain a fairly constant level of intracranial pressure in cases of tumor of the brain by dehydration and the balance of fluids, I think that if this were applied to the method of recording changes in the fields it might throw much light on the influence general intracranial pressure may have on changes in the visual fields and on scotomas.

Book Reviews

THE TREATMENT OF SCHIZOPHRENIA. By LELAND E. HINSIE. Price, $3. Pp. 206. Baltimore: Williams & Wilkins Company, 1930.

The author justifies the publication of this book by two reasons: (1) "the ever-increasing number of incipient schizophrenic patients who are seeking treatment," and (2) "the lack of a guide by which a clearer conception of the recognition and management of these cases may be had." He wants to overcome the medical hopelessness. He claims to be able to single out Bleuler's "latent schizophrenia" and "presents a single method of approach, that is, from the standpoint of psychopathology" — giving most of the space to an elementary consideration of fairly orthodox psychoanalysis and the rather incidental discussion of a few cases of as yet uncertain outcome. "At no time has it been our object to present the case material in detailed form, or to give a consecutive picture of the patient's psycho-physical evolution" (p. 77). "We selected only those patients who were still living in the surroundings natural to them. . . . We decided that each patient should be studied for at least one year, at regular intervals, for a total period of not less than one hundred hours" (p. 20).

The book opens with a general discussion of the emotions and ideas present in schizophrenia, "the views of others as regards, first, the emotions and, second, the thought content. The views appear to us to have some bearing upon the problems which are about to be discussed. We hope to be able to trace the genesis and course of the ideational and emotional characters in schizophrenic patients." He weighs W. W. Smith's associative method and then decides to use Freud's free associations "to discover whether what we call emotional deterioration is real or apparent, as measured by our present standards, the phrase being 'employed advisedly,' for at best the procedures that we possess do not lead to precise opinions. They may, however, be tentatively put into service for purposes of throwing some light on future methods." One wonders what the author has in store when he continues: "In the writer's opinion the Freudian scheme of pursuing investigations with the 'free association' method seemed to offer a suitable approach to the question at hand. We were not primarily interested in the matter of interpretation; indeed, for our present needs, we may leave 'interpretative psychology' out of consideration. After all, Freudian analysis is essentially 'resistance' analysis and does not consist of 'yardstick' interpretations." For the trained "analyst" the discussion offered of the method is at once too elementary and too diffuse, and a schizophrenic patient should not be made the victim of a beginner. The questionnaire of the mother-boy relationship (pp. 93-95) is difficult to harmonize with the principle of free association. The author evidently feels this (p. 99): "The physician best suited to carry on psychotherapeutic procedures is the one who has been successfully analyzed." He evidently does not shy from giving advice, but "ordinarily is it inadvisable to suggest marriage to the schizophrenic individual?" Is it ever advisable to suggest marriage? The chapter on the management of the sexual libido may well give cause for some questions as to the type of guidance and practical attitude or implications.

The chapter on physical symptoms contains two cases of head-feelings with definite transference "from below upward," i. e., pseudo-orgastic reaction in the head serving to delete the hallucinations or other symptoms.

The last chapter gives a review of methods of treatment in the form of non-critical brief abstracts of various communications.

The book seems rather hastily put together. The individual sentences, paragraphs and chapters (many of those already quoted) show this, purely from the angle of diction and logic, and from the angle of any mature consideration of the

statements. It is difficult to see how one can write chapter XII, the beginning of chapter I and the rest of the book, so to say, in the same breath and spirit. There is clearly a lack of inner cohesion and consistency and a fragmentariness by which perfectly good material and much good experience become decidedly weaknened through the lack of proper digestion. One can hardly speak of logic tight compartments, but much rather of nonlogical compartments, and many places where things are allowed to show confusion rather than mature fusion. Another edition will, the reviewer trusts, offer opportunities to incorporate the results of repeated perusal and discussion.

THE MOVEMENTS OF THE EYES IN READING. By M. D. VERNON. Special Report No. 148, from the Medical Research Council of Great Britain, July, 1930.

This pamphlet contains an account of work done by Miss Vernon for the Medical Research Council, and follows earlier work of 1929 (Report No. 130) on "Studies in the Psychology of Reading" by Miss M. D. Vernon and Mr. R. W. Pickford. The work was carried out in the Psychological Laboratory of the University of Cambridge. The report is divided into six parts:

1. The introduction, which mentions some of the important features of the study and the reason for them.

2. (*a*) The apparatus and its detailed description. In this a beam of light was reflected from the cornea to a moving camera film and a photographic record was thus obtained of the movements of the eyeball through the voluntary effort of reading. (*b*) Experimental material used. (*c*) The subjects of the experiments.

3. Results of the experiments on voluntary fixations and movements and a comparison of voluntary movements and fixations with return movements in reading. From the standpoint of physiologic optics and ocular movements, this section should be of great interest to the ophthalmologist. The marked differences in the accuracy of the subjects varied considerably with fixation and with the ocular movements. It was interesting that Miss Vernon discovered that on the whole the effects of fatigue and practice were very slight.

4. Results of the experiments in reading. From a psychologic standpoint, this subsection is not only valuable but interesting. It includes the methods of estimating the efficiency of the processes of reading, the effects of various mental processes on reading (illustrated by graphs), interest, associated thoughts and imagery (which were illustrated especially well by one of the subjects who was interested in the content of the reading material), the relationship of affective reactions to the nature of the process of reading and the various kinesthetic sensations accompanying reading. The last factor was checked carefully from the standpoint of four different factors that could control the development of these sensations. In fact, differences in the processes of reading are outlined by tables and by various graphs. In them the motor processes, the affective and conative reactions and individual interest were all considered. A summary of concomitant mental reactions to reading on the basis of these three probabilities was completed and is presented in a table.

5. The general conclusions to the summary of the work are of great interest. They include the following: "Thus it seems fairly clear that training and methods of learning to read play a large part in the formation of efficient habits of reading, and particularly of the average rate of reading and assimilation and of the suppression of tendencies to overrun the correct word. But the individual movements which make up the habit vary according to the motor ability of the eye muscles. And the rate of reading varies enormously according to a variety of mental processes, unconnected with training, which accompany reading, and of which conative impulses may possibly be the most fundamental."

6. In the concluding paragraphs, the subsections, as outlined, have again been abstracted from the standpoint of the results found and the conclusions drawn. The

pamphlet is readable and valuable, for it deals with the physiologic and psychologic aspects of the ocular movements and of ocular fixation. Being, therefore, the study of an important function and of the several factors that can and do modify this function, the reviewer feels justified in recommending it.

DEN FREUDSKE PSYKOANALYSE, DENS HISTORISKE BAKGRUNN. By RAGNAR VOGT. Price, 3.50 kroner. Pp. 101. Oslo: Gyldendal Vorsk Førlag, 1930.

In the last of a series of six lectures on psychoanalysis by a psychologist, three physicians and a theologian, the professor of psychiatry of the new psychiatric clinic of Oslo describes in his attractive and human manner the work of the forerunners of the latest of the "new" psychologies (reminding him of the "new logic" eclipsing that of Thomas Aquinas in 1330), the depth-psychology, which term of contrast should hardly imply a charge of superficiality against the insight of an Augustine, Kierkegaard or Nietzsche. Many currents ran together toward the end of the last century to create modern psychology, with psychoanalysis as one of the developments. Well chosen and lucid quotations anticipate the patterns of freudian terminology and their actual meanings, in a way reassuring one of a reasonable and natural evolution rather than of an abrupt burst of novelty sometimes claimed and widely assumed by a neophilic but historically poorly trained public. The wish, rationalization, substitution, Verschiebung (displacement), and condensation, the concern with love and hatred, the instincts, etc., are not so novel matters of concern as might seem to one not familiar with Locke, Hume, Feuerbach, Nietzsche, Lipps and many others; nor are the concept of the passive and the active components of mind, and the energetic dynamic approach, voluntarism, the unconscious, psychogenetic influences, regression, sublimation or Nietzsche's unmasking of symbols. The wealth of quotations is illuminating, and is not piled up to belittle what Freud has achieved, but to show the many bridges from the old to the more recent views. Ibsen naturally yields a wealth of illustrations and recognitions of what is perhaps too readily called "freudian mechanisms."

The whole presentation is free from any tendency to minimize the actual merits of the freudian synthesis of a personal system; but there is no evasion of the fact that the student of the past as well as of the present-day tendencies and developments may see how any sectarian success will yield and is already yielding to broader developments, which, moreover, need not all be psychologizing. Vogt wonders whether further broadening of what P. Briquet started in his book on hysteria in 1859 may not be the task or opportunity of physiology. The merit of the booklet lies in the interesting documentation and the thoroughly fair attitude. A translation would help many of the one-sided and poorly read sectarian devotees to a useful widening of the horizon, and save some of them from seeing in the freudian doctrine an overwhelmingly binding new philosophy.

DIE PFLEGE DER GEMÜTS- UND GEISTESKRANKEN. By W. MORGENTHALER. Im Auftrag der schweizerischen Gesellschaft für Psychiatrie. Price, 9.50 francs. Pp 273. Bern: Hans Huber, 1930.

The first part of the book (pages 1 to 56) deals with anatomy and physiology in a concise and simple but instructive form. More space is devoted to an explanation of the fundamental psychologic concepts and to practical psychology. The author uses an outline of a study of personality to determine the fitness of a person for the profession of nursing.

The second part (pages 57 to 114) explains the symptomatology and various reaction types, avoiding any far-reaching theoretical discussions.

Part three (pages 115 to 224) describes in detail the equipment of modern state hospitals and private institutions, how to observe patients, how to write reports and the care of a patient. In this part one finds excellent descriptions of how to deal with the various reactions. I do not think that any of the routine as well as the emergency procedures that nurses might be called on to use have

been forgotten. This chapter is also interesting and helpful to the psychiatrist. One misses, however, a discussion of packs. This is probably explained by the attitude of the German and Swiss psychiatrists, who still doubt their usefulness.

Part four (pages 225 to 246) presents social service, after-care and mental hygiene. It also refers to various Swiss laws with regard to mental care.

Part five (pages 247 to 256) contains a brief history of psychiatric treatment in general.

The appendix (pages 257 to 273) gives the rules that the Swiss Society for Psychiatry considers essential in the teaching of psychiatric nursing. Many illustrations of practical value make the reading easier and more interesting. Special care is given to the discussion of the prejudices against hospitals for the mentally defective and their treatment.

This book will be of great value not only to German but to American psychiatrists. Contributions of this type are still rare. The author has succeeded in writing a textbook for the actual management of mental patients and has avoided theoretical discussions whenever possible.

TREATMENT OF EPILEPSY. By FRITZ B. TALBOT, M.D. Price, $4. Pp. 308. New York: The Macmillan Company, 1930.

In the last two decades a great deal of research work has been done on the causation and treatment of epilepsy. Of the many books which have appeared, that of Lennox and Cobb, previously reviewed, is undoubtedly the best, for it presents in a concise and brief manner the scientific conception of the disease. It does not, however, discuss treatment at any length. This book serves that purpose. It has the added virtue of having received the helpful cooperation of Dr. Stanley Cobb and Dr. William Lennox.

The book contains about 300 pages. The first section of 119 pages gives a brief exposition of the etiology, pathology, diagnosis, prognosis, symptoms, prophylaxis and treatment. The description is just brief enough so that the student and general practitioner can get an adequate idea of the up-to-date points of view of epilepsy. Section 2, the balance of the book, discusses treatment. The subject matter is divided into chapters. It begins with a discussion of the factors necessary for an understanding of dietary treatment and is followed by a summary of the theories of dietetic treatment, the clinical results of fasting, the clinical results of the ketogenic diet, the practical uses of the ketogenic diet and the detailed management of the diet. In the author's experience the best results are to be obtained by the use of the ketogenic diet. His experience with dehydration has not been large, but the indications are that this method of treatment is difficult of application and not practical. In the hands of the author as well as other experienced neurologists the ketogenic diet scientifically applied promises the best results.

There is a valuable though brief chapter entitled "What to Teach an Epileptic Patient." However, throughout the text emphasis is placed on the nondietary aspects of treatment. The last chapter of half a page discusses migraine and merely mentions the occasional occurrence of migraine and epilepsy in the same patient or the occurrence of these diseases in families.

Altogether it is an excellent book and gives the best description of the detailed dietary treatment in print.